THE MEDICAL JOURNAL OF AUSTRALIA

VOL. I.—18TH YEAR. SYDNEY, SATURDAY, APRIL 4, 1931. No. 14.

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An Address.[1]

By GEORGE BELL, O.B.E., M.B., Ch.M. (Sydney), F.C.S.A.,

President of the New South Wales Branch of the British Medical Association.

THIS address marks the beginning of my year of office as President of the New South Wales Branch of the British Medical Association.

My first duty tonight is to express to you my appreciation of the honour you have conferred on me by electing me.

As you know, since 1930 the responsibility of delivering the presidential address has been assigned in New South Wales to the incoming President, whereas in the past the address was given by the retiring President. Does this imply that the address should be a forecast of the projected activities of the Branch during the coming year rather than a retrospective address? If the answer be in the affirmative, I think the task of the President has been made more difficult.

In my endeavour to discover the genesis of the presidential address I chanced upon the following interesting resolution which was passed at the original foundation meeting of the Provincial Medical and Surgical Association in 1832, which referred to the annual meeting:

> At this meeting it is proposed that one of the members shall be appointed to give at the next Annual Meeting an account of the state of progress of Medical Science during this year or an oration on some subject connected with medical science or a biographical memoir of some eminent cultivator of medical science who may have resided in the Provinces.

This retrospective address, as it came to be called, not invariably delivered by the President, is the

[1] Delivered at the annual meeting of the New South Wales Branch of the British Medical Association on March 19, 1931.

direct ancestor of the presidential address which has since been delivered annually.

In 1832 Charles Hastings founded at Worcester the Provincial Medical and Surgical Association and from this has grown the British Medical Association—British in the true sense of the word that today there exists in every part of the British Empire branches or bodies affiliated with it.

From the beginning the Association has had for its objects: (*a*) the promotion of the medical and allied sciences and (*b*) the maintenance of the honour and of the interests of the medical profession. It has been stated that the primary object of the British Medical Association is the promotion of the medical and allied sciences and I think that this is correct, for it is by promoting these objects that we can make the Association of the greatest benefit to the individual and to the State. We can thus follow the path already chosen for medicine—an idea so well expressed by Sir Arthur Keith:

> Given human nature as it is—a nature which demands and ever will demand that the poor must and shall have access so far as health is concerned to all the privileges that the rich can buy, that at all costs suffering must be relieved, thus medical progress must go on and follow the path already chosen.

I do not mean in any way to belittle the medico-political activities of the Association, but these must always remain of secondary importance. Should these activities ever become the primary object, we would richly merit the gibe sometimes applied to us, namely, that we are a trades union.

It is obvious that to attain this end the whole-hearted support of all types of practitioners of medicine is essential. How, then, can we best obtain the cooperation of all members of the Branch? The wide field of medical knowledge to be covered has rendered the formation of sections for the study of special branches of medical knowledge advisable. Thus we have in our Branch the Sections of: (i) Genito-Urinary and Venereal Diseases, (ii) Hygiene and Preventive Medicine, (iii) Medical Literature and History, (iv) Medicine, (v) Neurology and Psychiatry, (vi) Obstetrics and Gynæcology, (vii) Orthopædics, (viii) Oto-Rhino-Laryngology, (ix) Pædiatrics, (x) Pathology and Bacteriology, (xi) Radiology, (xii) Study of Cancer, (xiii) Surgery.

One sometimes hears the opinion expressed that the formation of these sections was a mistake and that the ordinary meetings of the Branch have not been so interesting nor instructive from the viewpoint of the general practitioner since their inception. Although there may be some justification for the above idea, I think the fault lies rather in a failure to carry out fully the original intention of the Branch. The formation of these sections has been a distinct advance and has followed the impetus given to specialism by those hand-maidens of medicine—the ancillary sciences, particularly in pathology, physiology, biology, physics and chemistry.

Now these branches of medical knowledge are all interdependent and have for their common object the pursuit of truth. By and large it is the function of the medical profession to apply this knowledge for the benefit of humanity for the prevention and cure of disease and the alleviation of suffering.

In recent times the advance of scientific knowledge in the fundamental and ancillary sciences has been followed by a corresponding advance in medicine. Specialism in the science and art of medicine has necessarily followed closely on this advance, and the great advance in all branches of medicine and the skilled technique required has made specialties most useful and beneficial—a true specialism built on a scientific foundation and not to be confused with what one may term "mushroom specialism" that grows up, as it were, in a night in response on the one hand to the demand of a generation seeking signs and wonders and on the other hand to a desire to acquire wealth rapidly. So long as the sections remain the servants and not the masters of the Association I am sure they will prove of great benefit to the Association.

With the short time at my disposal I propose to offer some constructive criticism and briefly to indicate how it may be possible for us more effectively to promote the primary object of the Association.

The Council.

While I think that the Councillors of the Association should be chosen from the very best men available, irrespective of the particular branch of medicine they practise, yet I am of the opinion that it is a matter for regret that the fundamental and ancillary sciences are not represented adequately on that body. I sometimes fear that if we neglect the primary objects we are apt to create a wide breach—a schism between the ancillary sciences—for instance, between pathology and the clinical branches of medicine. I would plead for a closer association of these sciences with the activities of the Branch, both in the council chamber and in the ordinary meetings. The representatives of these sciences should not hold themselves altogether aloof from the medico-political activities of the Association.

Pathology may, I think, be considered one of the greatest of the ancillary sciences, although I have heard the followers of this science described in jest as a "feeble folk." I am sure I am voicing the opinion of most clinicians and practitioners of medicine when I say that we regard them as the "giants" of the profession. It is they who provide the true foundations on which the best clinical work is built. There is a "pathology of the living"—a pathology of the quick as well as of the dead. We remember that it was on the science of bacteriology as practised by Pasteur that Lister based his work. How important, then, are they for the furthering of our primary object.

Meetings.

Ordinary Meetings.

The so-called ordinary meetings of the Branch constitute one of the best means of attaining our

object, for it is through these that we can best reach the great body of practising clinicians, the "men behind the guns"—the general practitioners—either directly or indirectly through the pages of our excellent MEDICAL JOURNAL OF AUSTRALIA. This being so, it behoves us to see that none but the best of work is presented at these gatherings. Quality, not quantity, should be our motto. It is an insult to the general practitioners to allow the "pot boiler" and advertiser to display his wares to them. Short shrift should be given to these folk. These meetings are for serious scientific discussion, not for personal advertisement. Prolixity should not be encouraged.

The practitioner of medicine is a busy man. This seems to be the day of many meetings. The activities of kindred medical societies in the large metropolitan hospitals and elsewhere have of late made further inroads into the leisure hours of our members.

For some time past I have often felt that there has been a diffusion and a wastage of energy in this sphere rather than a concentration of our activities in a few active societies. Moreover, all members of the profession can help to make these meetings a success. They should be a common meeting ground for all, both senior and junior members of the profession.

The Sections.

There is every indication that in the coming year the sections will do much towards providing interesting and instructive meetings. Nevertheless, the closest cooperation of all sections is needed.

While they should direct their attention to the closer study of their particular subjects, they should not become self-centred bodies, out of touch with the general body of the profession. Should they do so, they will tend to lose their sense of proportion. It is a matter for regret that those members who study the eye and diseases of that organ are not heard more frequently at the meetings of the Branch. The function of the sections should be to sift the wheat from the chaff and to present at the meetings the wheat and not the chaff.

The profession as a whole should play its part by attending these meetings in greater numbers and by their criticism help in the pursuit of truth and by testing the various hypotheses brought forward from time to time.

Clinical Meetings.

The fact that these meetings are so well attended proves that they are appreciated by the profession as a whole, and rightly so. They are essential to an effective study of the pathology of the living. So long as the clinical character of these meetings be maintained, that is, every facility given to members to examine the patients, they will be of great value. They should not be allowed to become chiefly meetings for discussion.

The so-called "open rounds" which are held at the various hospitals are in essence clinical meetings and therefore useful.

Sir William Osler has put the case for meetings very clearly:

> No class of men needs friction so much as physicians; no class gets less. The daily round of a busy practitioner tends to develop an egoism of a most intense kind to which there is no antidote. The few setbacks are forgotten, the mistakes are often buried and ten years of successful work tend to make a man touchy, dogmatic, intolerant of correction and abominably self-centred. To this mental attitude the medical society is the best corrective and a man misses a good part of his education who does not get knocked about a bit by his colleagues in discussions and criticisms.

Post-Graduate Work.

This is a more difficult subject. The selection of the appropriate measures for promoting post-graduate study is not an easy matter. The question is whether it should be carried out by the post-graduate committee alone or in conjunction with some other body which might be financially independent of the Branch, for example, a body composed of representatives from (i) the New South Wales Branch of the British Medical Association, (ii) the Faculty of Medicine, (iii) teaching hospitals. In any case, in order to insure success, a small committee of active minds is necessary.

I have purposely introduced the question of finance. Given a well arranged and good programme, a post-graduate course should be self-supporting, as it has been in the past.

Financial independence has been urged—perhaps to escape the oversight of the Executive and Finance Committee who, in these days of financial stress, are not inclined to sanction additional expenditure. Today this may entail some restrictions, but what of the future, when our building has reached a revenue-producing stage? Then, I have no doubt, not a little of the surplus funds will be devoted to the promotion of the primary objects of the Association, to post-graduate work and other schemes which promote the medical and allied sciences.

HODGKIN'S DISEASE.[1]

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FOREWORD.

To all workers attempting the study of Hodgkin's disease in Australia one great difficulty at the outset is the paucity of cases. The incidence per thousand in a world survey is small and naturally in our sparsely settled continent the work has been

[1] This work was carried out under the control of the Cancer Research Committee of the University of Sydney and with the aid of the Cancer Research and Treatment Fund.

delayed. Again, a diagnosis cannot be accepted on clinical findings alone. To differentiate the condition from many clinically similar complaints, careful and detailed histological evidence must be forthcoming. This involves removal of a gland and expert examination of the histological features displayed. We need consider only the numerous synonyms for the disease to realize the necessity for rigid diagnosis: (i) Progressive multiple lymph gland hypertrophy (Wunderlich),[69] (ii) lymphosarcoma (Virchow),[62] (iii) pseudoleuchæmia (Cohnheim),[10] (iv) *adénie* and *lymphadénie* (Trousseau),[59] (v) multiple lymphadenoma and leuchæmia (Wunderlich),[70] (vi) malignant lymphoma (Billroth),[4] (vii) chronic relapsing fever, (viii) lymphatic cachexia, (ix) lymphosarcomatosis, (x) chronic intermittent fever, (xi) multiple lymphomatosis (Korach), (xii) *lymphome ganglionnaire anémique* (Lanceraux), (xiii) myeloid sarcoma (Ziegler), (xiv) malignant granuloma (Benda and Fabian), (xv) lymphogranulomatosis (Benjamin and Grosz).

At the present time all histologists agree in accepting the standards of diagnosis enunciated by Sternberg,[56] Dorothy Reed[51] and Andrewes.[1] In our series of cases there has been rigid exclusion of any not exhibiting these standard histological features. Notwithstanding obstacles we have been very fortunate in selecting a fairly large number of patients. For this fairly constant supply of new cases we are indebted to many of our colleagues, most of whom have witnessed the results of the treatment later to be detailed.

In order to present a synopsis of published opinions and ideas relative to every aspect of the disease we are attempting a complete survey of the world's literature bearing on Hodgkin's disease published during the last hundred years.

Such research is necessarily time-consuming and so we intend to publish our work in three separate papers.

I. (*a*) History of the disease, (*b*) ætiology, (*c*) comparative pathology, (*d*) experimental investigation.

II. (*a*) Clinical signs, symptoms and sequelæ, (*b*) diagnosis, (*c*) differential diagnosis, (*d*) histology.

III. (*a*) Treatment, (*b*) prognosis, (*c*) serological treatment (research) carried on at Saint Vincent's Hospital.

It may not be premature to announce that we are inclined to favour the recently advanced suggestions that Hodgkin's disease is caused by a microorganism closely resembling the avian tubercle bacillus.

This is, in fact, the *raison d'être* of our work. After numerous experiments carried out at Saint Vincent's Hospital we were forced to agree with this hypothesis. A natural corollary was then the endeavour to procure a serum which might prove a guide to diagnosis because of a specific reaction in the patient. This we were unable to elaborate, but, whilst experimenting, we were surprised to find that a serum injection produced considerable diminution in size of the affected glands. Pursuing this line of research, we applied the serum as a therapeutic measure and this treatment has been adopted as a routine procedure in our cases. Results are being observed and all patients carefully followed up. Up to the present time no single patient whose condition has been diagnosed according to the standards enumerated above, has failed to respond in some manner to the serum injections.

We are waiting to observe results over a period of two years at least before reporting actual cures. During that period all recurrences or relapses will be recorded.

Deep X ray therapy has been withheld in nearly all cases. Obviously we realize that irradiation of the affected glands in lymphadenoma causes a rapid diminution in their size. This amelioration in the condition is by no means permanent. The glands enlarge again or new groups become involved, and the process, though delayed, ultimately means death to the patient. Experienced radiologists endorse this view.

As stated above, the detailed results of experiments, the method of preparation of the specific serum used and a complete record of cases will be published in our final paper.

Part I: History, Ætiology, Comparative Pathology, Experimental Investigation.

History of the Disease.

As in many outstanding discoveries in the medical world, the honour of classifying this disease as a clinical entity belongs to a worker in the fundamental sciences. Thomas Hodgkin,[28] Demonstrator in Morbid Anatomy at Guy's Hospital, London, in 1832 collected and studied seven cases. He epitomized his conclusions as regards the enlarged lymph glands in these words:

> As far as could be ascertained from observation or from what could be collected from the history of the cases, the enlargement of the glands appeared to be a primitive affection of these bodies rather than the result of an irritation propagated to them from some ulcerated surface or other inflamed textures through the medium of their inferent vessels, and that although in some instances the glands so enlarged may contain a little concrete inorganizable matter such as is known to result from what is called scrofulous inflammation, it is obvious that this circumstance is not an essential character, but rather accidental and concomitant to the idiopathic interstitial enlargement of the absorbent glandular structure throughout the body. That unless the word inflammation be allowed to have a more indefinite and loose meaning than is generally assigned to it, this affection of the glands can scarcely be attributed to that cause, since they are unattended by pain, heat and other ordinary symptom of inflammation and are not necessarily accompanied by any alteration in the cellular or surrounding structure and do not show any disposition to go on to the production of pus or any other acknowledged production of inflammation except where, as in cases above alluded to, inflammation may have supervened as an accidental infection of the hypertrophied structure.
>
> Notwithstanding the different characters which this enlargement may present, it appears in nearly all cases to consist of a somewhat uniform texture throughout and this rather to be the consequence of a general increase of every part of the gland than of a new structure

developed within it, pushing the original structure aside, as when ordinary tubercular matter is deposited in these bodies. At the time it must be admitted that the new material by which the enlargement is affected presents various degrees of organizability, which in some instances is extremely slight and appears incompetent to maintain the vitality of the affected gland.

Two of Hodgkin's original patients later were studied by both Symmers[58] and Dorothy Reed.[51] These workers, on histological grounds, proved conclusively the conditions were true lymphadenoma exhibiting the essential features still associated with the name of Dorothy Reed.

Hodgkin's original paper seemed destined to be cast into the limbo of forgotten things until in 1856 one of his students, Wilks,[66] drew attention to its value in a thesis entitled "Lardaceous Disease and Some Allied Conditions." In this he refers to Hodgkin's seven cases and, as a tribute to his teacher, labels them "Hodgkin's disease."

Virchow,[62] in 1864, described the condition and unconditionally classified it among the lymphosarcomata.

During this period numerous synonyms for the disease arose.

Cohnheim,[10] in 1865, refers to the malady as "pseudo-leuchæmia" and in the same year Trousseau[59] reported his series of cases as *"adénie."* A year later Wunderlich[70] (1866) adds to our list the resounding title *"Pseudoleukemia Hodgkinsche Krankheit oder Multiple Lymphadenoma ohne Leukemia."*

William Gowers[25] reviewed one hundred and fourteen recorded cases in 1879 and considerately refrained from bestowing another name.

Pepper's "System of Medicine"[47] in 1885 achieved some measure of immortality by including a masterly article on Hodgkin's disease by William Osler.

Pursuing our tortuous path, we now trace this clinical entity from the shadowy valleys of conjecture up to the well lit, broad ways of experimental and pathological evidence. Sternberg,[56] in 1899, studied the pathological histology of the affected glands and very definitely demonstrated that Hodgkin's disease must stand alone as a distinct malady. He accurately describes "changes in lymph nodes and elsewhere consisting in the appearance of a connective tissue reticulum supporting variable numbers of lymphocytes together with mononuclear and multinuclear giant cells with or without eosinophils."

Sternberg[56] studied fifteen cases and boldly states that all forms of glandular enlargement with recurrent pyrexia are tuberculous in origin. He claimed to have demonstrated this fact in ten of his cases and the remaining five are classified as being multiple lymphosarcoma.

The year 1902 saw established a firmly founded landmark in the study of Hodgkin's disease. Dorothy Reed,[51] of Johns Hopkins Hospital, and F. W. Andrewes,[1] of Saint Bartholomew's Hospital, London, in a series of cases carried out clinical, pathological and histological investigations. They then conclusively proved Hodgkin's disease to be a distinct clinical and pathological entity. Our present standards of histological diagnosis are directly attributable to these workers and, following their rigid requirements, we are enabled to classify our cases accurately at the present time.

Since that epochal year in the study of the disease many theses have been presented, but all workers agree that Sternberg, Reed and Andrewes have said the final word as regards diagnosis.

Ætiology.

We now approach a formidable barrier in our systematic study of Hodgkin's disease. What is the cause? Is it bacterial in origin or purely and solely neoplastic? This extremely debatable question of ætiology must now be surveyed and the different suggestions be recorded.

The problem is bristling with suppositions. Few diseases recognized at the present time can show such a wealth of conjecture as to ætiology.

Four main schools of thought may be recognized in this maze of theories, ideas and hopes, (i) atypical tuberculosis, probably due to the avian type, (ii) specific infective granuloma, (iii) new growth, (iv) a transition stage between granuloma and neoplasm.

Many investigators whose views cannot be lightly dismissed are at marked variance on these points.

Our experimental work inclines us in favour of the first of the above hypotheses, hence we regard the work of Elise l'Espérance[34] as of paramount importance.

Atypical Tuberculosis.

Confining our attention to the first school mentioned above, we find the names of many famous investigators coupled with the theory that the ætiological factor in the genesis of the malady is the tubercle bacillus.

Dickinson (1879) considered the disease allied to tuberculosis.

Sternberg (1899)[56] said it was a form of tuberculosis, dismissing those cases that did not conform as being multiple lymphosarcomata. He quotes thirteen cases of so-called "pseudoleuchæmia" which revealed tuberculosis, but his contemporaries, Westphal,[65] Dietrich[16] and Fischer,[21] were of the opinion that the tubercle bacillus was a secondary invader and, though possibly a factor in the production of the clinical picture, was not the sole agent. These three workers failed to isolate the tubercle bacillus and, their inoculation experiments not being successful, they naturally felt that they could not subscribe to the tuberculous theory alone.

Sternberg found disciples in Sailer[53] and Musser[42] who reported similar examples and reached the conclusion that the condition was that of a lymphatic tuberculosis.

Symmers,[58] in an excellent paper, which is referred to later, shows:

> Hodgkin's disease maintains its histological individuality throughout all changes of environments. This, if anything, favours inflammation against neoplasm.

Fagge,[20] in 1874, and again Delafield,[13] in 1905, described a primary glandular Hodgkin's disease that clinically could not be distinguished from tuberculosis.

Weber,[63] who is justly famed for his clinical observations and who will be the subject of reference later, stated that the lesions of Hodgkin's disease were favourable soil for tuberculosis and that children with Hodgkin's disease ultimately developed fatal disseminated miliary tuberculosis. Andrewes,[1] defending his aversion to the tuberculous theory, reminds his auditors that the lymphatic glands act as filters to all microorganisms and the mere presence of tubercle bacilli does not mean that the particular lymph gland is necessarily infected or invaded by tuberculosis. Against this Pizzini,[48] to show the widespread occurrence of the tubercle bacillus, states that of all persons dying of Hodgkin's disease 42% showed the presence of tubercle bacilli. The tracheo-bronchial lymph glands gave the highest percentage of positive findings. Northrup,[45] in fact, found the tubercle bacillus limited to the bronchial glands in thirteen out of one hundred and twenty-five children who died of tuberculosis.

Holt and Loomis[37] are investigators who also support the tuberculous theory in the ætiology of Hodgkin's disease. Quincke[50] (1902), however, says that tuberculous infection in cases of Hodgkin's disease often causes some amelioration of the symptoms, but the mere fact of finding tubercle bacilli in an enlarged gland does not necessarily mean that the original lymphadenomatous condition should be classified as Hodgkin's disease.

In Dorothy Reed's published series of eight cases only one patient died of tuberculosis. No tubercle bacilli were found in the other seven. All animal inoculation experiments failed to produce tuberculosis.

Wuttke[71] states that Hodgkin's disease is caused by a modified strain of the tubercle bacillus. Loygne[38] holds that the cause is in the antrum and is most likely the tubercle bacillus. Finally, Symmers[58] gives as his considered opinion:

> Hodgkin's disease is neither infectious nor neoplastic, but a systemic disease expressing a predilection for lymphoid tissue giving rise to multiple foci of growth at approximately the same time in response to the same provocative agent.

Before leaving the intriguing hypothesis of a tuberculous origin for Hodgkin's disease, we should like to report a part of Sir H. Butlin's address[8] before the Royal Society of Medicine. On the occasion of Andrewes's epoch-making address on the history of Hodgkin's disease Butlin said:

> In the first place the disease which we have been talking about is certainly not tuberculosis nor does it render a patient in the least degree immune to tuberculosis and I should like to go a step further and say I have a suspicion that lymphadenoma renders a patient on the whole more disposed or liable to an attack of tuberculosis than he would be if he were not suffering from this particular disease.

Specific Infective Granuloma.

Continuing our survey of the main schools of thought regarding ætiology, we now consider the hypothesis that the tumour masses of Hodgkin's disease are really specific infective granulomata.

The evidence brought forward by the proponents of this theory is slender and very conflicting when we consider the comparatively large number of its adherents who have undertaken investigations.

In 1892 Dreschfeld[17] described a bacillus which he asserted was found in the lesions of all patients. Again, in 1895 Delbet[14] claimed to have isolated the causative bacillus which on animal inoculation reproduced the disease. Stengel[55] advances a total of twenty-nine cases in which he discovered microorganisms.

The perusal of the literature again brings us to the year 1902 when, as previously mentioned, Reed and Andrewes established a definite landmark in the study of the disease.

McNalty,[41] basing his work in the definite pathological and histological features elucidated by Reed and Andrewes, investigated the question of ætiology once again and in 1911 summarized his findings as follows:

1. Blood films were all negative as regards the presence of parasites.
2. Examination of material obtained by splenic puncture failed to disclose any microorganisms.
3. Blood cultures were all sterile.
4. Exhaustive tests of the various human secretions failed to yield any causative microorganism.

Fischer,[21] in a review of twelve cases, finds all cultures and animal inoculations to be without result, except in one instance where "organisms were present in the blood during pyrexia and absent at all other times." Then, unfortunately, he leaves us still benighted. The nature of the microorganism is not stated. He finally suggests the pyrexia itself may have been due to a secondary infection by these organisms and not an integral part of the Hodgkin's syndrome.

The year 1917 saw a serious effort to place the specific infective granuloma theory on a sound footing. Bunting and Yates[7] published a thesis asserting that there was a common ætiological factor in leuchæmia, pseudoleuchæmia and Hodgkin's disease, these diseases being merely varied reactions to a similar stimulus. Should this stimulus be a toxin, then the milder doses would cause stimulation and hyperplasia as in the lymphoblastic and lymphocytic groups. The presence of the same toxin in large amount would destroy normal structure as in Hodgkin's disease.

From the enlarged lymph glands in all these conditions they claimed to have isolated a Gram-positive pleomorphic diphtheroid bacillus.

They record the finding of the organisms in all their cases, but quite frankly they themselves question whether they have any real significance as regards ætiology. Against their supposition it was reasonably argued that the very diversity of clinical conditions in which the organism was supposed to appear, itself negatived the possibility of any ætiological importance being attached to it. The actual experimental work of Bunting and Yates will be referred to later. These diphtheroids of Bunting and Yates bear a close resemblance to those discovered by E. de Negri and Mieremet,[44] who labelled them "corynebacteria." Fraenkel and Much[23] claimed to have isolated a similar organism as the causative factor in Hodgkin's disease.

Arrayed against the theories of Bunting and Yates we find Weber[64] and McNalty[41] who were unable to confirm the bacteriological findings. Also Rolleston[52] remarks that their evidence is not conclusive, as the early hyperplastic changes in human lymph glands supposedly due to their "toxin" is merely the ordinary inflammatory reaction of lymph glandular tissue.

Longcope,[36] Cunningham and McAlpin,[12] together with Stewart and Dobson,[57] support Rolleston, referring to a "peculiar giant cell reaction, probably the foreign body type of Bunting and Yates."

Twort,[60] whose work will be referred to later, found guinea-pigs to give merely an inflammatory reaction to the injection of lymphoid tissue.

P. F. MacGinnis,[40] Pathologist to the Chesterfield and North Derbyshire Royal Hospital, writes:

> We have found a large spirochæte in the cervical glands of two consecutive cases of Hodgkin's disease and we believe this to be an important etiological factor in the disease.

He goes on to say that a detailed account of the facts will be published later.

Nakamura[43] (1916) carefully examined six cases of Hodgkin's disease and concluded that the clinical and the histological factors more closely resemble those of a chronic infective process than of a neoplastic process. He points out that there is no proof of a true metastasis in Hodgkin's disease, but there is a pseudo-metastasis, that is, the enlargement of distant lymph glands which more closely resembles syphilitic lymphadenitis than neoplastic disease.

Nakamura found antiformin-fast rods in tumours, lymph glands and spleen in three of his cases. He further demonstrates the close relationship of histological features of primary disease of the lymphatic apparatus, especially affecting the endothelial cells, and contrasts the histology of lymphosarcomatosis and leucosarcomatosis. He compiled the appended table which enumerates the various conditions affecting the lymphatic system. This gives an excellent picture of the diseases resulting in alteration of the lymphocyte balance of the gland in comparison with those which result in alteration of the endothelial cell balance.

I. Hyperplasia of the Lymphatic Cell.
   1. Lymphatic leuchæmia.
   2. Aleuchæmic hyperplasia or pseudoleuchæmia (myeloma and Mikulicz's disease).
      (*a*) Regional form.
      (*b*) Generalized form.

II. Hyperplasia of the Reticulo-Endothelial Cell.
   1. Hodgkin's disease or malignant granuloma.
   2. Tuberculosis.
      (*a*) Slightly caseating form.
      (*b*) Non-caseating, a diffuse cell hyperplasia.
   3. Syphilis.

III. True Tumour.

New Growth.

Are the enlarged matted glands of Hodgkin's disease purely neoplastic in origin? So many skilled observers incline to this view that we must give it due consideration.

Bright[6] observed:

> There was another form of the disease which appears to be malignant in character, though it varies from the more usual form of malignant disease, and which has been particularly pointed out by Dr. Hodgkin as connected with extreme disease of the absorbent glands, more particularly those accompanying the blood vessels. The whole of these absorbent glands become larger and firmer with tendency to suppurate, as in ordinary scrofulous disease or to soften as in cerebriform disease, at the same time.

Markham's dictum was that:

> There was little evidence to show that they were malignant and not stronger than they were scrofulous.

Letulle, Trémolières and Moussoir,[35] in their "*Étude d'un cas de maladie de Hodgkin*," support the new growth theory of ætiology. In their case nearly all the organs contained nodules and after minute microscopical study they state that since Sternberg cells were found in a "metastatic progression" outside the blood vessels and outside the limits of the nodules, that is, in the surrounding healthy tissue, the condition must necessarily be malignant in nature.

Ewing's[19] ideas on this aspect of the ætiology may be summarized as follows:

1. Extreme degrees of endothelial hyperplasia are not infrequently associated with and dependent upon granulomatous infiltration of lymph nodes. Such cases demonstrate the capacity of endothelium to respond to inflammatory irritation with extensive proliferation.

2. In some cases it is difficult or even impossible to determine whether this overgrowth is simply inflammatory or whether it is independent of the irritant being autonomous and neoplastic.

3. The long continued effects of inflammatory infiltration may lead to neoplastic growth of lymphatic endothelium. In the course of such inflammatory infiltration of lymph nodes after repeated operation the granulomatous element may be eliminated and the disease progress as a neoplasm.

4. Granulomatous infection of lymph nodes in a very early case gives rise to excessive overgrowth of endothelium of a distinctly anaplastic type and with local invasive properties.

5. Such malignant endotheliomata may arise apart from any evidence of the previously associated granuloma. It is possible to conceive that an original granulomatous area may be overgrown and obscured by neoplastic cells. Of this no definite evidence has been secured nor has it been shown that one node of a chain may exhibit purely neoplastic overgrowth whilst other nodes are granulomatous.

6. Certain endotheliomata of lymph nodes, variously designated as diffuse flexiform alveolar or perivascular, are probably derived from the endothelium of lymph sinuses and lymph cords.

7. Certain primary tumours of lymph nodes, with or without associated granulomata, are probably derived from the reticulum cells of the follicles. The tumours resemble lymphosarcomata with large cells and may be distinguished from tumours composed of small lymphocytes.

8. Endotheliomata of lymph nodes differ from the strictly classified new growths in several particulars and may be regarded as a disease *sui generis*, although essentially a neoplasm.

Kundrat[32] asserts that he has observed cases of lymphosarcoma arise from pseudoleuchæmia and from Hodgkin's disease.

These theories, especially those set out by Ewing, will be referred to later when our original observations will be discussed.

A Transitional Stage Between Granuloma and New Growth.

The fourth theory listed above has proved attractive to many competent observers. Apparently the conflicting evidence, some in favour of specific infective granuloma, some equally convincing as regards a neoplastic origin of Hodgkin's disease, has led to the acceptance of this assumption.

Ewing appears, in his published work, to be distinctly in accord with this hypothesis, and if the salient points of his views, tabulated above, are recalled, we cannot fail to recognize a strong bias towards this decidedly easy way out.

Dreschfeld[17] studied two cases and examined the excised tumours for microorganisms with negative results. He divides the disease into acute and chronic forms—the former he calls lymphosarcomatosis, calling it an infection of the specific type which may follow a local focus or may be general from the onset. The chronic type he subdivides into: (*a*) Like the acute form, only chronic in its course; (*b*) a form of leuchæmia, that is to say, a hyperplasia of the blood-forming organs, becoming a lymphatic, splenic or myelogenous type, though the latter is not yet clearly established.

He summarizes his ideas as follows:

I have come to the conclusion that there is no fundamental structural difference between a lymphadenoma and a lymphosarcoma. Such differences as are noticed as regards structure can be easily explained by the rates of growth, the preponderance of certain embryonic cells, and so on, and that clinically also the two forms must be looked upon as identical.

Yet, despite this, he goes on to state that he considers the disease due to a specific infective agent. His ideas regarding the cause of sarcoma cannot be taken too seriously, as he wrote this article in 1892.

Cherry,[9] in his interesting work, "The Tubercle Bacillus and Cancer in Mice," draws attention to the similarity between leuchæmia, pseudoleuchæmia and lymphosarcoma, and quotes Simond's work, which indicates that there is no hard and fast line between pseudoleuchæmia and sarcoma in mice. Cherry also refers to the evidence connecting lymphosarcoma with tuberculoma in animals and man, and goes on to say that Hodgkin's disease is related to tuberculoma on one hand and sarcoma on the other. In his series of mice inoculated with tubercle bacilli Cherry states that there is no line of demarcation between the enlarged glands and the lymphosarcomata and both are equally involved in the lymphoid syndrome, but that the lymphosarcomata are not ordinary manifestations of tuberculosis. In conclusion he says that the change to malignancy might be some intermediate step in this lymphoid syndrome if it be conceded that leuchæmia is a condition related to malignant disease.

Longcope,[36] of the Ayer Clinic in 1903, states:

Hodgkin's disease has been considered by many as a malady of obscure origin, which stands rather as a clinical condition than as both a clinical and pathological entity. And it is doubtless for this reason that lymphatic tuberculosis, lymphosarcoma and other diseases which produce enlargement of the lymph nodes have from time to time been confused with true Hodgkin's disease.

Also that:

No definite predisposing causes are known, though both Winewarter[27] and Gowers[26] mention local irritation as a possible factor. In the majority of cases there is no family history of syphilis or tuberculosis. Throat and eye troubles or sometimes deafness may precede the glandular swelling. Indeed, it has been suggested that the tonsils may offer a road to the entrance of infection, but this view does not seem to be upheld by anatomical findings, for it is in fact comparatively rare to see these organs involved early, though the tonsils and even the gums may become the secondary growths. In none of these present cases were the tonsils diseased.

Comparative Pathology.

In a search through the literature regarding the comparative pathology aspect, one is surprised to note the appalling lack of cooperation between the veterinarians and the medical investigators. The small amount of work that has been done on animals is sadly marred by the lack of histological evidence to support claims made.

Sir John McFadyean[39] collected five cases in lower animals, but prefaced his thesis, and largely negatived it from our viewpoint, by saying:

If the Reed-Andrewes histological features be essential for the diagnosis of Hodgkin's disease, then the cases under discussion had no title to be called lymphadenoma at all. They must perforce be styled lymphoma.

Hodgson[29] described a somewhat similar syndrome in a pig, but found the glands on section to show an excess of lymphocytes instead of endothelial hyperplasia and Reed cells.

Woolridge[68] states that Hodgkin's disease is rare in animals. Clinically the dog shows a somewhat similar syndrome, but histologically the results are entirely negative, only lymphocytic hyperplasia being demonstrable. This type of disease occurred in a bull which, under treatment by an arsenical preparation and cod liver oil, recovered so completely that a buyer later paid £1,000 for the animal. Whether he was given the full veterinary history Wooldridge does not state!

Sir H. Morley Fletcher[22] quotes cases in which improvement with arsenic has occurred. He suggests this disease in animals may be infective in

origin and cites the possibility of a spirochætal invader. Lymphosarcoma superimposed on Hodgkin's disease caused him to wonder, he states, in reporting such a case, whether the sarcoma commenced in the gland or in the surrounding tissues.

Leslie Pugh[49] lets a ray of light shine on the veterinary aspect of the question by being definite at least. No single case of Hodgkin's disease corresponding to the Reed-Andrewes pathological requirements has been proven in animals. He has never heard of the Pel-Ebstein syndrome in veterinary literature and very aptly questions whether temperature charts were ever kept throughout the progress of these cases.

Ellerman[18] states that in transmitting leucosis in fowls a number of his subjects developed Hodgkin's disease. This statement cannot be accepted since none of the tissue examined showed the Reed-Andrewes histological picture. The pathological changes seen were of the nature of a true lymphocytic hyperplasia.

W. H. Andrews[2] (1926) states bluntly that if one accepts the Reed-Andrewes pathological picture for the diagnosis, then not a single case is reported in veterinary literature. Until more is known of the ætiology of Hodgkin's disease, errors will be made and possibly authentic cases missed. A particular microorganism might cause in animals of different species a reaction varying in clinical features, but essentially identical.

H. H. Scott[54] (1925) Prosector of the Zoological Society, refers to the disease in monkeys as follows:

> The animals are marmosets (*Callithrix jacchus*). One was left at the gardens on account of a swelling in the neck. It died in a few days and the autopsy revealed enlarged glands on both sides of the neck, in the groins and thorax. The thymus gland was enlarged and the spleen was twice the normal size. Glands on section showed the typical histological features of Hodgkin's disease. In addition to the glandular increase of eosinophils there was definite eosinophilia in the peripheral blood. This may have been due to the presence of *Filaria gracilis*, but no parasites were found in the glands.

McFadyean[39] (Royal Veterinary College) diagnosed Hodgkin's disease in four dogs at autopsy and in one clinically. Guinea-pigs were inoculated with the material obtained. These were healthy on being killed four months later. The glands on section failed to reveal any trace of the Reed-Andrewes pathological appearances, except that the distinction between medulla and cortex of the gland was totally abolished. He recognizes these appearances differ from tuberculosis and by exclusion is inclined to classify them as Hodgkin's disease.

The work of M. H. Gordon[24] (1925) who uses guinea-pigs, was aimed at proving a spirochætal origin for Hodgkin's disease. His results were all negative. Although he failed to isolate spirochætes, it does not necessarily follow they are totally absent (*vide* Sobernheim in Kolle and Wassermann's text book). Gordon describes his technique for growing spirochætes and strongly advises anaerobic cultures. He urges other workers to proceed along the lines that had proved successful in the search for *Spirochæta pallida*.

It is interesting to note that such eminent veterinarians as Hutyra and Marek,[30] in their text book (1926), refer to the same difficulties as regards ætiology in animals as we admit in man. They discuss the possibilities of tuberculosis, of new growth, of a specific infective microorganism.

We found the researches of these men with the avian tubercle bacillus interesting and stimulating in view of our own opinions as regards the ætiological factor in Hodgkin's disease. Pursuing investigations with chickens, they elucidated the fact that true leuchæmia in veterinary medicine is exclusively avian. This disease may be transmitted to chickens by intravenous and intraperitoneal inoculations of affected organs. Other birds and animals are not susceptible. About 40% of the chickens develop the disease in one or two months after inoculation. Skibia, however, considers chicken leuchæmia to be different from the leuchæmia of man in that the chicken variety is merely an intense leucocytic reaction to infection or trauma. Chickens exhibit a marked leucocytosis in trivial infections.

Hutyra and Marek describe a syndrome in horses, dogs, cattle and cats which they describe as Hodgkin's disease. They consider that other workers have confused this disease with leuchæmia exclusive to chickens and missed the correct diagnosis.

Experimental Investigation.

The close relationship of the anthropoid apes to man in the evolutionary scale led Cunningham and McAlpin[12] to choose these animals for experiments in 1923. All previous experimental investigation had been performed on the lower animals. Their work was in essence an attempt to prove their own hypothesis that Hodgkin's disease was protozoal in origin.

Four experiments were made, three *Macacus rhesus* and one bonnet monkey being used. The possibility of these animals being tuberculous was excluded by guinea-pig inoculation of secretions.

Experiment 1: A piece of lymph gland showing the histological picture of Hodgkin's disease was implanted in the ileo-caecal mesenteric glands. The animal made a good recovery, but nineteen weeks later died accidentally owing to an overdose of chloroform. At autopsy a number of enlarged lymph glands were found in the mesentery. One at the site of implantation showed an abscess cavity lined by a palisade of large multinucleated "giant cells" intermingled with lymphocytes, plasma cells and polymorphonuclear pus cells. The "giant cells" were of the "foreign body" type, however, neither the tubercle bacillus nor any other organism was demonstrable.

Experiment 2: This experiment was a failure, as the monkey, a male rhesus, had to be killed on the tenth day, on account of a suppurating wound.

Experiment 3: Male rhesus. An emulsion of a lymph gland showing Hodgkin's disease was injected (by syringe) into the spleen and into two large glands in the ileo-caecal angle. Laparotomy one year later disclosed extensive adhesions around the spleen. A large nodule, one quarter of an inch in diameter, found in a lymph gland proved to be an abscess containing a nematode. This same monkey, on being killed two years later, failed to show any abnormality at autopsy.

Experiment 4: A coarse emulsion of a Hodgkin's gland was injected into the spleen and lymph glands of a male

bonnet monkey. In addition a few chips of the diseased gland were implanted in another lymph gland. Four and a half months later the spleen was excised. No enlarged lymph glands were seen; the spleen proved to be normal. During the operation a large amount of material from the same patient with Hodgkin's disease was implanted into lymph glands, into the abdominal wall and under the rectus muscle. The ape, when killed two years and eight months later, failed to reveal any abnormality whatever at autopsy.

Cunningham and McAlpin summarized their results as follows:

> These experiments afford further proof that whether or not Hodgkin's disease is an infective granuloma it is not due at least to a virus capable of reproducing the disease in monkeys.

Attention must be focused on the work of Twort and Craig.[61] Their paper, "The Pathogenicity of Johne's Bacillus Compared with that of Other Acid-Fast Bacilli for Some of the Laboratory Animals," proved particularly interesting in view of its agreement with our own conviction that Hodgkin's disease is caused by a tubercle bacillus variant—probably the avian type.

The first description of a disease later to be known as Johne's disease was the work of Johne and Frothingham,[31] who recognized the syndrome in a cow. These workers believed an avian type of tubercle bacillus was responsible. Twort and Craig, in their paper referred to above, mention that Johne's disease has been described as occurring only in cattle, sheep and deer.

Bearing in mind our record of previous investigators of Hodgkin's disease, their series of experiments are of peculiar interest. Having made subcutaneous injections of Johne's bacilli into rabbits, they produced a caseous abscess at the site of inoculation. This persisted for a long period. They were impressed by the resistance to autolysis shown by the large numbers of bacilli in these lesions. Although the bacilli died rapidly (cultures taken a week after inoculation proved sterile), their bodies are not disintegrated for many months; they remain well formed and capable of full differential staining. When these dead bacilli were reinjected, they showed the same resistance to disintegration. Various internal organs of the rabbits used failed to show any bacillary invasion.

They next inoculated several hens intravenously with an emulsion of Johne's bacilli. The conditions noticed in the rabbits were reproduced almost identically. Multiple doses of *Bacillus phlei* were next injected into another series of hens. These at autopsy revealed multiple nodules in the liver and spleen, some of which attained the size of a bean. Histologically these nodules showed a typical picture of necrosis with acid-fast bacilli present in large numbers. In other hens which lived and were killed many months later, all organs were free of bacilli and demonstrable lesions.

Twort also found that guinea-pigs frequently reacted to inoculations of pieces of lymphoid tissue with the production of local transient nodules which histologically were inflammatory.

Bunting and Yates[7] consider that a Gram-positive pleomorphic diphtheroid bacillus was the cause of Hodgkin's disease. This has been referred to above. Over a period of five years they made cultures from the neck, groin and axillary glands of their subjects and proved the presence of these diphtheroids. Another series of their experiments claims notice. Inoculating monkeys, dogs, rabbits, horses and guinea-pigs with Hodgkin's disease material, they produced abscesses at the point of inoculation and lymph gland changes "similar to those seen in early Hodgkin's disease." They do not specify these changes, however. They also claimed to have effected a deviation of complement in the patient's serum by using these diphtheroid bacilli as antigen. However, they finally concluded the record of their experiments with the admission:

> Such evidence would be accepted as of ætiological significance for almost any organism except for members of the diphtheroid family, but the final test, that is, the production of a disease in animals comparable to the human type, has not been accomplished.

Billings and Rosenow[3] (1913), Hatcher and Lemmon[27] (1915), Bloomfield[5] (1915), Harris and Wade[26] (1915), Olitsky[46] (1915) and Cunningham[11] (1917) performed experiments in connexion with the diphtheroid theory of Bunting and Yates. All finally disputed the theories advanced, but failed to record any constructive research.

Stewart and Dobson[57] (1924) grafted a small piece of Hodgkin's disease gland on to the mesenteric glands in thirty-nine monkeys, and in two of them an emulsion was also injected into the spleen. An inflammatory reaction occurred, exhibiting large foreign body giant cells and a simple lymphoid hyperplasia. Monkeys injected intravenously also yielded negative results.

Fraenkel and Much,[23] in 1910, undertook to confirm or disprove Sternberg's invaluable contribution to the study of Hodgkin's disease. This attempt involved a most complete and intensely interesting series of experiments. In the account of their researches they describe the "antiformin" method of isolating microorganisms from the enlarged glands of Hodgkin's disease. By the use of this they isolated heavy and fine granular rods which occurred in clumps. Photographic illustrations lend credence to this claim. Their results were confirmed experimentally by other authors. Rabinowitch, however, failed to find these acid-fast organisms in a repetition of the experiments. Possibly the failure was due to the use of Hodgkin's glands not sufficiently enlarged and mature.

In 1913 Fraenkel and Much carried out animal experiments on a large scale. Monkeys, rabbits, guinea-pigs and dogs were used. Into the peritoneal cavities of these animals was injected an emulsion of Hodgkin's disease glands previously treated with antiformin. The particular glands used were excised from patients proved to be suffering from Hodgkin's disease and in whom all possibility of tuberculosis had been rigidly excluded. Some of the guinea-pigs

died and tuberculous lesions were demonstrable at autopsy in spleen, lungs and substernal lymph glands.

Then several series of experiments with adequate controls were performed on guinea-pigs exclusively. Some animals received minced glands alone; others minced glands previously treated with antiformin. Another batch were injected with minced glands previously treated with lactic acid which had been credited by French authorities with the ability to enhance the virulence of microorganisms. Approximately one half of the injected animals died with typical lesions of tuberculosis. Considering a differential review of their results, Fraenkel and Much concluded that the antiformin treatment of the glands resulted in a diminution of the virulence in the organisms concerned, whilst the lactic treatment served to enhance the bacterial virulence. So using lactic acid only in the previous treatment of the glands, they injected another series of guinea-pigs. Curiously enough, the animals appeared to maintain normal health. Glands removed at autopsy after the animals were killed were introduced into other guinea pigs. These animals developed tuberculous lesions. Still further experiments in which the use of lactic acid was discarded, resulted in the production of tuberculous lesions in lungs, liver and spleen of 50% of the animals used after an incubation period of five months. Fraenkel and Much, confining their attention to the organism responsible for these phenomena, definitely label it a tubercle bacillus, though admittedly of a peculiar strain. They studied it morphologically and culturally.

Negri and Mièremet,[44] also in 1913, inoculated guinea-pigs, but their results were negative, no tuberculous lesions being observed. Using spleen and lymph glands, they also injected three mice subcutaneously, one rabbit intraperitoneally and one monkey subcutaneously. Two weeks had elapsed when the monkey developed a tumour at the site of inoculation. This gradually disappeared, but two months later enlarged glands were palpable in both groins and in both axillæ. These were excised. Cultures on different media and under varying conditions were made. In tubes to which spleen pulp had been added successful growth was obtained. This growth proved to be composed of a pure culture of granular, acid-fast rods similar to those previously described by Fraenkel and Much. Other culture media showed a culture of fine rods with polar staining and on a bile medium cocco-bacilli were grown. The granular rods subcultured on Loeffler's serum produced a profuse growth of comma-shaped rods.

Animals were inoculated with these cultures with rather conflicting results. Smears were made from the slightly enlarged lymph glands, later excised, and the granular bacilli were again seen. No macroscopical result (cultural), however, was obtained except on Loeffler's serum. On this medium a corynebacterium, morphologically identical with the granular rods described by Fraenkel and Much, was obtained. Negri and Mièremet hence conclude that the causal organism of Hodgkin's disease is this corynebacterium. Complement deviation tests failed when patients' serum and this organism were used.

This work raised a storm of comment and argument. Koch and others questioned whether this corynebacterium exists in animal lymph glands and whether its isolation and cultivation in these experiments was sufficient proof that human lymphadenoma is actually caused by this microorganism.

Fraenkel and Much now reviewed their earlier work in the light of subsequent evidence. They consider that Hodgkin's disease is caused by the close staining rods described by themselves a peculiar and rare type of tubercle bacillus. In a subsequent work they discuss the clinical aspect of the disease so produced and consider that Hodgkin's disease is a rare tuberculous disorder, but not rarer than tuberculous lupus and other similar conditions. They say it is probably the result of exhausted lymph glands responding to a tuberculous infection which only differs slightly from a pulmonary tuberculosis.

No new departure in medicine nor any discovery in science has sprung full fashioned from the research of any groups of workers. In every instance the slow, toilsome climb may be traced until at the peak we find that the fortunate one adds the final stone to the monument.

The gradual culmination of experimental inquiry as to the ætiology of Hodgkin's disease leads to the work of Elise l'Espérance,[34] from the Department of Pathology of Cornell University, New York, United States of America. The result of a series of most important experiments was submitted for publication in June, 1928. The inspiration for our own efforts has been directly derived from this publication; so important did we find this work that the author's own words are quoted here:

Condensed Protocols.

In the first experiment two white Plymouth Rock chickens were inoculated with a typical Hodgkin's node obtained from the New York Hospital. One bird died five months after inoculation with lesions involving the liver and spleen. These lesions consisted of focal granulomata composed of many large polyhedral cells, myeloid giant cells and eosinophiles with more or less extensive necrosis.

The second chicken of this series died eleven months after injection with similar lesions in liver and spleen and the added involvement of the lymphoid structures. The histology differed from the first one in that the necrotic foci appeared larger and more numerous, occasionally fusing to form conglomerate tubercles or abscesses.

Direct smears from the nodules from both of these chickens showed acid-fast granules, extra- and intracellular. Cultures from one of them on Dorset egg media gave a scanty growth of acid-fast bacilli with contaminating blastomyces. On account of the presence of acid-fast bacilli these cultures were reinoculated into other chickens, but the time is too short to determine the results.

In the second experiment three Plymouth Rock chickens were inoculated with a typical Hodgkin's node obtained from the Memorial Hospital.

One of these chickens died seven weeks after injection with an enormously dilated, congenitally deformed heart. The liver and spleen contained many small white foci

quite distinct from the parenchyma. These foci proved to be collections of large polyhedral cells, round cells and giant cells without necrosis. Material from these small tubercle-like lesions in the liver and spleen was reinjected into normal Plymouth Rock chickens. One of these reinjected chickens died eleven months after inoculations with an abdominal mass, about the size of a small orange, in the region of the rectal fold. This mass was composed of a conglomeration of nodes, discrete, yet bound together, encircling the intestine and attached to the mesentery. There were isolated firm nodes scattered through the mesentery and many firm nodules in the cervical region. The liver, spleen and bone marrow also contained tubercle-like structures.

On section the large mass appeared firm, greyish in colour, without necrosis and each node was quite distinct from the other.

On microscopic examination the lesions were found to consist of large round polyhedral cells, many myeloid giant cells, lymphocytes and eosinophiles in granulomatous arrangement.

Direct smears from the nodes of neck and abdomen stained by Ziehl-Nielsen method revealed acid-fast bacilli.

Of the original three chickens of this series a second one died seven months after inoculation. The autopsy showed a large liver studded with small opaque areas, and at the base of the skull, firmly attached to the bone and dura and adherent to the brain, there was a small, hard nodule, well circumscribed, but not encapsulated. The histology of this brain nodule and the liver foci was identical with that observed in the other two chickens of this series—a diffuse granuloma with little necrosis and many myeloid giant cells.

A sensitized guinea-pig, inoculated with material from the liver nodules from this chicken, died with an atypical tuberculosis nine weeks after the injection. The histological picture of the lesions was characteristic of a granuloma with little necrosis, many myeloid giant cells and fibrosis. Cultures from the nodes of this pig gave moist, slightly yellowish growth of acid-fast bacilli, suggesting the avian type of tubercle culture.

The third chicken of this group was killed twelve months after injection and revealed scattered foci in liver and spleen, and an unusual hyperplasia of lymphoid tissue in the cervical region.

Histologically the lesions showed the characteristic proliferation of large cells and giant cells.

Together with these experiments we append the author's own summary and her deductions from the results obtained.

Five healthy chickens were inoculated intravenously with emulsions of lymph nodes from two cases of clinically and histologically characteristic Hodgkin's disease.

All of these chickens developed either a typical or an atypical tuberculosis, and in the tissue smears stained by Ziehl-Nielsen method, acid-fast granules and rods, extra- and intracellular, were demonstrated in three of them, and non-acid-fast granules in one of them.

Reinoculation of material from the lesions of one of these chickens into another chicken gave an identical, though more extensive, manifestation of the disease.

An atypical tuberculosis developed in guinea-pigs by inoculation of the tissue from the fourth chicken. A growth of bacteria with the staining and cultural characteristics of the avian tubercle bacillus was produced on egg media from material from this guinea-pig.

Cultures from these birds on egg media were contaminated in all instances. In one the chicken culture gave a symbiotic growth of blastomyces-like organism with an acid-fast bacillus. These cultures were reinoculated into normal chickens and guinea-pigs, but sufficient time has not elapsed to determine the results of this reinoculation.

In a second communication Elise l'Espérance conclusively demonstrates the reason why other workers had consistently failed to inoculate guinea-pigs with Hodgkin's disease.

This animal is notoriously resistant to infection by the avian strain of tubercle bacillus; but l'Espérance succeeded in producing lesions by previously "sensitizing" the guinea-pig by means of injection of a dead heterologous strain of tubercle bacillus, bovine or human. This paper will be referred to of necessity in our later communications regarding our own work.

In January, 1929, a further investigation was done by De Leon and Reyes[15] along the lines of interest stimulated by l'Espérance's work.

On May 7, 1925, two monkeys were engrafted from the same patient (P.G.H. 53-2, 1925) and on July 26, 1928, one monkey was engrafted from another patient (P.G.H. 53-4, 1928), both of whom had been clinically and histologically diagnosed to be suffering from Hodgkin's disease. The material consisted of Hodgkin's nodes from the left side of the neck and the right groin, which were grafted among the lymph nodes in the groins, and in the abdominal fascia and mesentery of the animals—whole and encapsulated nodes in monkey number 1, cut nodes in monkey number 2 were used in monkey number 3, the material for the last mentioned monkey having been obtained from the second patient's right inguinal lymph nodes. This animal was injected intraperitoneally with an emulsion from another piece of tumour tissue which had been previously macerated. As much as possible the grafts were implanted on the portion where the capsule of the lymph nodes had been incised, so as to allow the closest contact between native and engrafted tissues and thus facilitate invasion and subsequent growth. The monkeys all did well after operation. Their wounds healed. No abnormal changes were noted in their appetite and behaviour. On palpating the more superficial grafts after one month no masses could be felt except the operative scars. There were no growths of any kind. The first two animals were killed after a month, but at autopsy showed no new growths or evidence of the grafted tissues. They evidently absorbed all the implanted growths without harm to themselves. The third animal is still alive and is being kept under observation.

Thus far, therefore, we have no evidence of the implantability of Hodgkin's disease in such experimental animals as dogs.

In this connexion it may be illuminating to recall some of the results of l'Esperance's experimental inoculation of chickens with these nodes; for instance, the development in the chickens of typic and atypic tuberculosis and the subsequent cultivation of avian tubercle bacillus from material obtained from a guinea-pig which had been previously inoculated from one of the chickens. Do our negative results in monkeys and l'Esperance's positive results in animals admittedly susceptible to tuberculosis lend support to the belief, held by some, that tuberculosis and so-called Hodgkin's disease are identical, or at least closely related? May not the latter disease really be nothing more than a modified form (glandular form) of tuberculosis?

Acknowledgements.

Before concluding the first section of our paper we should like to express our thanks to those who have personally helped us. First, to our colleagues who have referred patients to us; to Dr. F. P. Sandes, Director of Cancer Treatment, University of Sydney, for his personal interest and monetary assistance; to Mr. Tunnicliffe, of the Fisher Library, University of Sydney, who so generously devoted an enormous amount of time in selecting and searching the literature; to Mr. Caminer, who so cheerfully volunteered to help with the translations from German; and to Mr. R. Walker, whose assistance in the laboratory with the chickens and with the

patients under treatment has been most valuable and who also expressed his willingness to help with the clerical compilation and statistics involved in this work; and, lastly, to Dr. A. J. Fitzgerald, who has given greatly appreciated editorial assistance.

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Reports of Cases.

PRIMARY INTUSSUSCEPTION OF THE APPENDIX.

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Primary intussusception[1] or inversion of the appendix itself is rare, but Battle and Corner[2] have succeeded in collecting seventeen cases from the literature.

The following description of a case which was encountered recently by the writer, may therefore be of interest.

W.C.H., a well nourished boy, aged eight years, was admitted to the Newcastle Hospital on October 11, 1930, complaining of abdominal pain. His mother stated that on the day prior to admission he had been seized with a severe pain in the middle of the "stomach," had vomited once and then felt better. Next morning a slight pain was still present, but he appeared better and had no further vomiting. Two hours later the pain returned with increased intensity and vomiting recommenced. He was then seen by Dr. J. R. Leslie and sent into hospital with a diagnosis of acute appendicitis. His bowels had not been open since the onset of the attack. He had had no urinary symptoms or other complaints.

The boy was seen to be in pain. He lay recumbent, with the right leg drawn up and seemed disinclined to have the abdomen touched. The abdominal wall was not rigid, but was very tender to palpation in the right iliac fossa. There was no palpable mass present. An examination *per rectum* revealed nothing abnormal. The other systems were free from any abnormal clinical signs.

Operation.

Under open ether anæsthesia the appendiceal region was approached by the paramedian route. The caecum was located higher up than usual, about the level of the umbilicus, and it was found to be involved in a mass which was delivered easily through the wound after a little manipulation. It was then possible to examine the anatomical relationships thoroughly. The ileum entered the caecum in the normal manner and was not involved in the mass, which consisted of an œdematous portion on the antero-medial aspect of the caecum and an intussusception at the base of the appendix (see diagram).

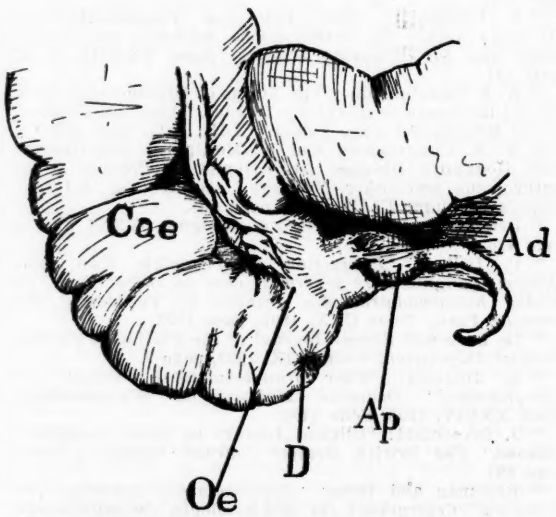

Diagram of Specimen. Cae = caecum; Oe = œdema; D = dimple; Ap = appendix; Ad = adhesion.

The base of the appendix was abnormally wide and at the point where the intussusceptum entered it, the appendiceal wall was inflamed and swollen into an œdematous, spherical mass. The distal portion of the appendix was normal. By grasping the caecum and freeing the recent adhesions binding down the swollen portion of the intussusceptum it was possible to "milk" the parts back into their normal relationship, thus revealing a very long appendix with a wide, flabby base. The appendix was excised, the stump invaginated and the bowel restored to the abdominal cavity.

The abdominal wall was sutured in layers and the patient made an uneventful recovery, leaving hospital on the fourteenth day after the operation.

Comment.

A macroscopic examination of the inner wall of the appendix subsequent to the operation revealed an area of gangrenous mucosa situated about 4·0 centimetres distal

to the base.. This seemed to indicate that an acute appendicitis had been the initial factor in bringing about the irritation and increased peristalsis necessary to produce the intussusception.

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EPITHELIOMA OF THE PENIS TREATED BY RADIUM.

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AND

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REPORT OF CASE.

(W. MAXWELL.)

J.B., a male, aged seventy-three years, while being treated for diverticulosis of the sigmoid colon, complained of irritation of the prepuce and *glans penis*. Examination on November 5, 1929, revealed a mild chronic balanitis with thickening of a tight redundant prepuce. Mild antiseptic baths were given for immediate relief, and circumcision was advised.

Six weeks later the patient complained of increasing difficulty in retracting the prepuce and stated that a tender "wart" had made its appearance. Examination revealed increased thickening of the prepuce with granulations and purulent exudate on its deep surface. On the dorsal aspect toward the left side was a flattened cauliflower-like lesion about the size of a threepenny piece, situated on the fornix of the prepuce and extending on to the corona and *glans penis*. The tumour was indurated and commencing to ulcerate. The glands of the groins were palpable, but it was thought that they were clinically normal or mildly inflammatory. The diagnosis of squamous-celled carcinoma was confirmed by biopsy. There was no reaction to the Wassermann test.

Retraction of the prepuce was difficult and painful, so a dorsal slit was made on January 4, 1930, with local anæsthesia. Dr. Moran was consulted and with his cooperation and material radium treatment by a surface method was instituted, applications being for ten hours daily over a period of eighteen days. A mould of Colombia paste 1·5 centimetres in thickness was made to ensheath the whole length of the penis, the proximal end resting upon the pubes and fixed in position by means of a strap passed around the pelvis. Thirty-five milligrammes of radium element in seven foci were disposed around the circumference of the mould at the level of the lesion, the long axes of the tubes being parallel to each other. For filtration a layer of platinum 0·5 millimetre in thickness was employed. The testes and neighbouring parts were protected by lead sheeting. At no time during treatment was there any disturbance of micturition.

The radium reaction had disappeared at the end of six weeks, the epithelioma at the end of three weeks. Circumcision with local anæsthesia was carried out immediately after the subsidence of the reaction. The wound healed very slowly.

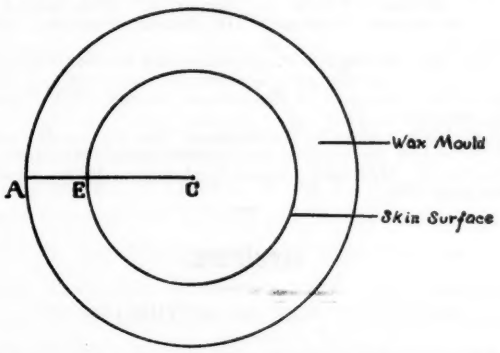
The glands of the groins received no treatment whatever. To date there is no evidence of metastases.

COMMENT.

(H. M. MORAN.)

Epithelioma of the penis is stated by Ewing[1] to form 1% to 3% of all cancers in the male. Phimosis, syphilis and leucoplakia are accepted as causative factors. There is an almost complete absence of the disease in the Jewish race. Of 75 cases recorded by Dean[4] there was phimosis present in 74, in 20 a history of syphilis, and in three signs of leucoplakia. On the other hand, it is said that in China, despite the absence of the factor of phimosis, this new growth occurs and has been ascribed to the use of severe irritants in the topical treatment of venereal disease.[3] "The established disease regularly takes the form of a papillary epithelioma and may long remain a relatively warty tumour of considerable dimensions, but ulceration commonly supervenes . . . Papillary tumours of the glans are nearly always malignant" (Ewing.[1])

The neoplasm is of comparatively slow growth. Glandular metastases may reach a large size, but in the early stages the enlarged inguinal glands may be inflammatory. There has always been a strong prejudice on the part of the patients against an operation which demands amputation. The results from the early use of deep X radiation, however, were not satisfactory, nor were those obtained by the embedding of radium needles. Dean[4] in 1926 published a statistical examination of 64 cases in which radium was applied locally with deep X rays for the inguinal glands. Le Roy des Barres, Ferrari and Viallet, Murdoch and Loicq followed with similar reports, and Bodescu[5] in 1928 published his observations on eleven cases at the *Institut du Radium* at Paris. In passing it is to be noted that this is a relatively rare form of malignant disease in France.



Le Roy des Barres[7] established clearly the superiority of a technique which makes use of a mould in Colombia paste. This is commonly of a thickness of 1·5 centimetres. The cylindrical form of the organ makes it easy to realize a correct and homogeneous irradiation.

The appreciation of the value of the time factor in the treatment of squamous epithelioma leads us to spread the exposure over a minimum of four days. The method used for Dr. Maxwell's patient was based on that described in Réchou and Wangermez's[6] article in the *Archives d'Electricité Médicale*. For example, if we take the organ as a cylinder, the radius of which is 2·5 centimetres, and apply a wax mould with a thickness of 1·5 centimetre, it is easy to work out the depth dose.

In such a case AE in the diagram = 1·5 centimetres, AC = 4·0 centimetres, EC = 2·5 centimetres. The relation of the doses at the skin surface E and at the centre of the organ C would be:

$$\frac{(AE)^2}{(AC)^2} = \frac{(1{\cdot}5)^2}{(4)^2} = \frac{2{\cdot}25}{16} = \frac{1}{7} \text{ approximately.}$$

Thus it would be necessary to place seven sources of radium circumferentially around the organ at equal distances apart. The perimeter is $2\pi r = 2 \times 3{\cdot}14 \times 4 = 25$ centimetres approximately. The sources would therefore need to be some 3·5 centimetres distant from one another. It can be verified mathematically that the dose obtained at any spot on the surface of the organ is equal to that at E or C. The actual dose to be given was worked out by Réchou and Wangermez with an ionomicrometer. In their case, for a surface of 200 square centimetres, 200 milligrammes of radium element used for one hundred hours were found to give a radio-epidermitis curable in four weeks. It might be better to extend the treatment over a longer period. With a filter consisting of one milli-

metre thickness of platinum at a distance of 1·5 centimetres a dose of 0·75 millicurie destroyed per square centimetre of surface can be given.

It is necessary to protect the testes by heavy layers of lead and by separating the parts as widely as possible.

The problem of the glands has next to be considered. In early cases without signs of glandular involvement it is permissible to adopt a policy of watching and waiting. But the mere suspicion of glandular metastases demands surgical excision followed later by deep X radiation or by radium application at a distance.

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Reviews.

MALIGNANT DISEASE OF THE LARYNX.

THE Anglo-French Library of Medical and Biological Science is to be commended upon the issuing of the monograph on cancer of the larynx.[1] We feel that it is right that this subject should be considered in book form by such an eminent man in this branch of surgery as is Sir St. Clair Thomson. There are many surgeons who have been following his work and his teachings in journals for a number of years and have lamented the fact that these have not been made available before in a complete form. To these practitioners especially we feel sure that the book will be very welcome. We feel, too, that it is only proper that this subject, the advancement of which in the past has owed so much to British workers, should now be brought up to date by the leading British representative of the specialty. The choice of Mr. Lionel Colledge as a cowriter is a good one, for he represents the younger British laryngologists who have shown that they are also competent to undertake treatment of extrinsic laryngeal and pharyngeal cancer.

The subject has been treated in detail from all aspects and the book is copiously illustrated.

Throughout the chapters on signs and symptoms the slow growth and relative innocence of the intrinsic form, and the more malignant characters of the extrinsic variety are constantly being emphasized. Particularly do they point out that "every adult with hoarseness should be considered as possibly cancerous until proved otherwise" (a dictum of Chevalier Jackson), and the book under review, we hope, will do much to draw the attention of the general practitioner to this most important symptom. The useful classification of intrinsic, extrinsic and subglottic cancer has been adopted and it is emphasized that "there is no internal region of the body where cancer gives such an early and decided warning of its occurrence as in the cavity of the larynx where it remains so long localized." The opinion is expressed that surgeons should refrain from performing a preliminary diagnostic biopsy, except in cases of doubtful diagnosis, for fear of stimulating, while not removing, the cancerous growth by local trauma. They also consider that a positive biopsy should be followed by a radical operation. Immobility of the vocal cord is stated to be a symptom of advanced laryngeal cancer. In the chapter on the operation of laryngo-fissure it is definitely stated that a satisfactory approach and removal require the excision of one ala of the thyreoid cartilage. This is a very important part of the operative technique and is not appreciated by some surgeons. Sir St. Clair Thomson's results are based upon 70 cases which show 76% of lasting cures, which is probably the best series of lasting cures of any surgeon, past or present. In laryngectomy the authors prefer a one stage operation and consider a preliminary tracheotomy a disadvantage unless some special indications are present. They emphasize the need of attention to mouth and nose sepsis. The description given of local anæsthesia indicates a not very extensive use of the method by the authors, therefore they rightly prefer to use general anæsthesia, and they strongly urge against all preliminary medication by drugs. For obvious reasons we do not approve of the suggestion that the complementary tube of the suction apparatus be employed to blow the chloroform vapour down the larynx and trachea. In the section of pharyngotomy, the lateral transthyreoid, the medium translingual and the subhyoid methods of approach of Trotter are well described and are illustrated by Trotter's own drawings. Based upon Trotter's opinion, they consider that the statement that extrinsic cancer involving the pharynx is hopeless and not amenable to treatment is too sweeping. The section on the lymphatic system of the larynx is illustrated by two excellent coloured pictures by Hajek, and the surgery of the invaded glands by two coloured illustrations by Crile. The risks of treatment by X ray and radium radiation are emphasized and the methods of application are briefly touched upon, and it is stated that "unless radiation can show results as lasting as those that have been demonstrated to be obtainable by surgery, it should be reserved for cases with indications which render them unsuitable for surgery."

We consider that this excellent volume should be possessed by every specialist in laryngology and we recommend it also to the general surgeon and to the general practitioner.

[1] 'Cancer of the Larynx," by Sir St. Clair Thomson, M.D., F.R.C.S., F.R.C.P., and Lionel Colledge, M.B., F.R.C.S.; 1930. London: Kegan Paul, Trench, Trubner and Company Limited. Demy 8vo., pp. 266, with illustrations. Price: 25s. net.

RADIOLOGY IN THEORY AND APPLICATION.

AN interesting account of the generation and physical properties of X rays is given by Terrill and Ulrey in their work on X ray technology.[1]

Though the book is primarily written for the laboratory investigator, there is much in it of value to the medical radiologist. The practical details of the construction of X ray tubes, the different types and operating characteristics of the latter, methods of high voltage generation and rectification, and other problems and difficulties of X ray engineering are matters that concern all radiologists and ones with which they should be more familiar than many of them are; whilst the various chapters on the physical measurements of voltage and current, ionization, total energy and wave length respectively, and that on biological measurements are of particular importance to those practising X ray therapy.

The statement generally is scientifically and commendably concise and is eminently readable, except, perhaps, in the final chapter on the X ray analysis of matter, dealing with emission spectra and the structure of crystals, where brevity has been carried to the point of sketchiness.

[1] "X-Ray Technology, the Production, Measurement and Applications of X-Rays," by H. M. Terrill, Ph.D., and C. T. Ulrey, Ph.D.; 1930. London: Chapman and Hall. Royal 8vo., pp. 264, with illustrations. Price: 21s. net.

The Medical Journal of Australia

SATURDAY, APRIL 4, 1931.

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BRANCH COUNCILS.

A SCIENTIFIC society, like sections of a community and like the community itself, must have an executive body to organize its activities, to superintend its corporate acts so that they will conform to the provisions of its constitution, and to keep a watchful eye on individual members so that they do not in their enthusiasm wander beyond their legitimate bounds on the one hand, nor on the other grow weary in well doing from laziness or procrastination. The British Medical Association is no exception. The parent body in England has its President and its other office bearers, including its Council. The members of the Association in the United Kingdom are grouped into Divisions and Branches; and the Australian Branches have the status of Branch-Divisions. Each of the Australian Branches is represented on the Council in England and each has its own office bearers and its council, elected periodically by its members. The office of councillor is no sinecure. The work to be undertaken, especially in the larger Australian Branches, is assuming alarming proportions and promises to increase. Not only have the scientific activities of the Branch to be outlined for each period of twelve months; the finance of the Branch has to be taken in hand, ethical matters have to be arranged and disputes settled, matters of contract practice have to be discussed and decisions which may be far reaching have to be arrived at, problems under *Workers' Compensation Acts* have to be attacked and other questions have to be faced.

It is quite obvious that all members of a Branch have not the qualities which will allow them to deal effectively with all these matters. Some, for example, will be more suited to give advice in connexion with the acceptance of papers from the point of view of their scientific value; others again, from their personal experience, will be able to offer prompt and sound advice on the matter of contract practice. In nominating members of a Branch for election to a position on the Council members should bear this in mind. They must not necessarily choose the man with most leisure. Leisure is often synonymous with laziness. It is the experience of most officials of medical organizations that the man with the busiest practice is the man who has most time to spare—he is keen and, by apportioning his time, he can devote attention to many subjects. A council should therefore include representatives from all branches of the medical profession: general practitioners, physicians, specialists in diseases of particular systems of the body, and those who devote their whole time to the basal sciences and to teaching. It is this point which is the logical corollary of Dr. George Bell's remarks in his President's address published in this issue. His appeal is opportune.

Complaints and murmurs of dissatisfaction are not infrequently heard about the management of the affairs of the Branches. It is our firm conviction that these grumblings are more often than not born of ignorance of the nature and magnitude of the problems faced by the Council. It must be remembered, when dissatisfaction is expressed, that the Council of a Branch has been appointed by the members of the Branch—each member has the same voting power—and that the members who have appointed one body of councillors, may, if they choose, appoint a new council at the following election. It is well also to draw attention to the reluctance on the part of many men to allow their names

to be submitted to a vote. It is a healthy sign to see a large list of nominations on a ballot paper, provided the candidates are genuine in their desire to promote the welfare of the Branch. Some persons may become candidates for council honours because they are rebels. If there are such, they might well be appointed; they would probably have their eyes opened at the first meeting. Dr. Bell has appealed for wide representation on the Council of his Branch; if this is desirable in the New South Wales Branch, it is also desirable in the Branches of other States. He has referred more particularly to practitioners who devote their lives to the study of the ancillary sciences; they are probably the most reluctant to seek nomination and on account of their special knowledge they could undoubtedly give material help in guiding the scientific endeavours of the Branch. His words will, we hope, bear fruit; but not only this group of practitioners, but every other group must take its share of the work. The profession in a State cannot be effective unless it is united, and union may be brought about by a continual striving by all the members to uphold the traditions and further the objects of the British Medical Association.

Current Comment.

INTESTINAL OBSTRUCTION.

In what manner does acute intestinal obstruction cause death? The answer to this question has not yet been supplied. Dehydration of the body, inspissation of the blood, starvation, the action of toxins produced by putrefaction of the intestinal contents above the constriction, shock due to the sudden trauma to the intestinal nerves *et cetera* have all been suggested as direct or contributing causes of death. Within recent years Williams declared that *Bacillus welchii* proliferated in the intestinal tract and produced a toxin which had lethal effects on the organism. The results he obtained by the use of *Bacillus welchii* antitoxin encouraged others to work along similar lines, and now his theory is widely accepted. Whipple's theory is that a toxæmia is due to the absorption of a proteose formed in the mucous membrane of the intestine itself.

An individual affected with starvation or dehydration alone does not die within so short a period as that within which death usually takes place as a result of acute intestinal obstruction. There must be some other factor or factors. The development of a toxin of some sort is the first possibility which comes to mind. Conceivably toxins may arise from bacteria, devitalized tissues or putrefying food, and may be so powerful as to endanger life. To each single theory of the cause of death from intestinal obstruction there are objections. It seems probable that there is a combination of several factors—not necessarily always the same combination—whatever the cause of the obstruction and whatever its site.

The results of experimental studies on intestinal obstruction in dogs have recently been published by J. C. Armour, T. G. Brown, D. M. Dunlop, T. C. Mitchell, H. H. Searls and C. P. Stewart.[1] The objects of their investigations were to determine the cause of death following obstruction in the proximal portion of the small intestine and to devise a means of preventing death.

In the consideration of the effects of intestinal obstruction a distinction must be drawn between simple constriction and strangulation or kindred conditions. A loop of intestine deprived of blood supply dies and is thus not only itself a source of poison, but also it is a medium eminently suitable for the proliferation of various organisms. Furthermore, organisms are likely to escape from such a section of gut, to infect other tissues, notably the peritoneum. Armour and his collaborators wisely confined their experiments to simple obstruction.

These workers remark that, if any of the various theories of toxæmia be correct, there should be greater toxæmia, hence earlier death, when the constriction is low in the intestine, as there is then a greater area of bowel for the absorption of the toxin. But it is known that death ensues most early when the obstruction is just below the pylorus.

Recently the theory has been advanced that death is due to alteration in the chemical composition of the blood; it is said that dehydration, alkalosis and loss of chlorides occur. The administration of saline solution has been advised by several workers. White and Fender succeeded in keeping a dog alive by reinjecting the gastric contents into an enterostomy opening below the obstruction. Armour and his collaborators conducted their experiments with the object of investigating the blood chemistry as well as making a study of the bacteriology of the intestinal contents.

Six dogs were used in the experiments which apparently were carefully conducted and adequately controlled. The intestine was divided at about the level of the tail of the pancreas. The upper segment of bowel was occluded in approved fashion and returned to the abdominal cavity, the end of the lower segment was fixed to the parietes and fitted with an enterostomy tube. No food or water was administered by the mouth as a rule; water was given occasionally for the purpose of washing out the mouth or stomach, but it was always withdrawn immediately by means of a stomach tube. Vomiting

[1] *The British Journal of Surgery*, January, 1931.

was prevented as far as possible by withdrawing the gastric contents several times a day. Food was administered through the enterostomy tube. Three dogs received no treatment and succumbed a few days after operation, despite artificial feeding. In one of these three the biliary and pancreatic ducts were anastomosed into the intestine below the enterostomy; the results in this instance differed in no particular from those in the other two. This is interpreted by the investigators as convincing evidence against the theory that a toxin is formed from the bile or pancreatic juice. In each instance the carbon dioxide combining power of the blood rose rapidly from 55 or so to 95 or 110 volumes *per centum* before death. On the other hand, the blood chloride content fell in an equally striking manner; this was accompanied by a corresponding fall in the quantity of sodium chloride in the urine.

To three other dogs saline solution, in addition to food, was administered through the enterostomy tube. The weight of these dogs gradually diminished and it was evident that adequate nourishment could not be supplied through the enterostomy opening, but the carbon dioxide combining power and the blood chloride content remained at about the normal level so long as the saline solution was administered. One dog was kept alive and well for a period of six weeks; the administration of saline solution was then discontinued, with the result that the blood chloride content fell rapidly, symptoms of alkalosis, as lethargy, twitching and weakness, ensued, and the animal died after typical *grand mal* convulsions. Another dog died of an infection of the upper part of the respiratory tract. The remaining dog was treated for six weeks; the administration of saline solution was then stopped, with the clinical and chemical results observed in the other experiments. To save the animal's life saline solution was again administered and, within a few days, the continuity of the gut was restored by means of operation. At the time of operation the segment of gut above the obstruction was found dilated and hypertrophied, evidence in the opinion of the investigators that there had been here a collection of stagnant material suitable for the growth of bacteria. This, however, may be regarded as a debatable question; in the first place there is only circumstantial evidence that a collection of stagnant fluid existed for any length of time and, assuming the existence of such a collection of fluid, as no examination of its composition could be made, who may say whether it was favourable to bacterial growth or actually inimical to it?

It is of interest to note that in no instance was there any great diminution in the quantity of chlorides in the gastric contents, thus great quantities of chlorides were lost to the body in the vomitus. Often there was no evidence of free hydrochloric acid. Armour and his fellow investigators remark that there was apparently little diminution in the quantity of hydrochloric acid secreted, but that it was immediately neutralized by mucins or the regurgitated contents of the duodenum.

Bacillus welchii was found similarly in the stomach contents of treated and untreated dogs. The investigators conclude that this bacillus cannot be regarded as a factor in causing death. It is worthy of note that some specimens contained no *Bacillus welchii*, while others from the same animal did. From a study of the urine and blood no evidence of excessive protein destruction in the tissues was discovered.

The authors are commendably conservative in their discussion of their results. They do not claim that they have established the validity of the chemical theory, nor that they have absolutely disproved the intoxication theory, but they believe that they have collected almost conclusive evidence in these directions. They admit the possibility that there might be some other factor the ill effects of which are prevented by the action of the saline solution. This aspect is of great interest; other workers have noted that pyloric obstruction may cause alkalosis owing to the impossibility of reabsorption by the intestine of chlorides secreted by the stomach. If the obstruction be lower in the intestinal tract, though the stomach contents are persistently evacuated by vomiting, it is reasonable to believe that some sodium chloride does escape through the pyloric orifice into the intestine, whence it is reabsorbed into the blood stream. Possibly enough chloride is thus retained in the body to postpone death a little longer than when the obstruction is high in the intestinal tract. This is not incompatible with the intoxication theory, for, though the toxin may be less when the obstruction is more proximal, there is then also a more rapid loss of chloride and a more pronounced dehydration, so that there is less material for the dilution and neutralization of the toxin. This is pure theory, of course, but indicates that a solution has not yet been reached.

It may be asked why does persistent vomiting now cause an acidosis and now an alkalosis? In the one instance ketosis is due to the breaking down of the body's nutritional reserves consequent upon starvation, in the other there is an excessive loss of chlorides, hence alkalosis. But surely the same breaking down process takes place in this instance also. Is the acid loss really greater than the ketone gain or are there fallacies in the available methods of chemical investigation of the blood?

This work is an important contribution to medical knowledge. Its chief value lies in the hope it affords for the successful treatment of individuals affected with obstruction of the small intestine. A study of the body metabolism following obstruction in the lower part of the ileum should provide useful information if conducted on similar lines to the investigations of Armour and his coworkers. While all due acknowledgements should be given to these investigators on their excellent work, it must be admitted that much remains to be done before the effects of intestinal obstruction are satisfactorily understood.

Abstracts from Current Medical Literature.

DERMATOLOGY.

Acrodermatitis Continua and Psoriasis Pustulosa.

H. W. Barber (*The British Journal of Dermatology and Syphilis*, November, 1930) discusses *acrodermatitis continua vel perstans* (*dermatitis repens*) and *psoriasis pustulosa*. He points out that many so-called cases of the former are really examples of the latter. He tabulates the chief differences between the two conditions. Some of the characteristics of pustular psoriasis are as follows. It may begin bilaterally, there is seldom any history of injury and it is often symmetrical with a predilection for the thenar eminences and the inner sides of the insteps. The nails are as a rule unaffected, but when they are involved the lesions are non-suppurative and resemble those of ordinary psoriasis. There are no mucous membrane lesions. The patches begin as reddened areas in which pinhead-sized pustules form; most of these dry up to form minute brownish scabs. Attempts at bacteriological culture are as a rule without result. The opposite conditions generally prevail in *acrodermatitis continua*. In conclusion, the author states that the condition described by Crocker as *dermatitis repens* is identical with the *acrodermatitis continua* of Hallopeau and may be conveniently described as *acrodermatitis perstans*. He maintains that it is a clinical entity and must be differentiated from exematoid ringworm, moniliasis, epidermophitides, infective dermatitis and psoriasis. The onset and evolution of the eruption and results of bacteriological and serological investigations point to the view that it is an invasion of the skin by a strain of *Staphylococcus pyogenes aureus* possessing distinctive activities in that it grows freely on media that as a rule inhibit staphylococcal growth.

Purpura Annularis Telangiectodes.

R. Curle and S. W. Smith (*Edinburgh Medical Journal*, November, 1930) report a further case of *purpura annularis telangiectodes* in a woman, aged sixty-five years. The disease is usually described as occurring in three stages: (i) Telangiectasia, (ii) hæmorrhage and pigmentation, (iii) atrophy. It is characterized by a ringed pattern and slow progress. It started in the case reported as an eruption of tiny reddish purple rings on the legs, thighs and lower part of the abdomen. There was no discomfort from it. There was a history of menstruation up to sixty-four years of age. The patient noticed a marked thinning of the hair. There were no pregnancies, no history of rheumatism or Bright's disease and no familial tendencies. The skin generally was congested and the extremities livid. After seven months the rash had spread over most of the trunk and portions of the limbs. The rash was bilaterally symmetrical. An interesting feature of the case was the presence of a large fibroid tumour about the size of a four months' pregnancy. After operation the rash started to fade. The trouble seemed to be caused by a venous congestion, the result of a vasomotor paresis brought about by some anti-intoxicant acting on the walls of the terminal capillaries of the viscera as well as the skin.

Eczema.

P. Escudero (*Revue Sud-Américaine de Médicine et de Chirürgie*, November, 1930) discusses occult diabetes and eczema. He regards eczema as local or symptomatic (*l'eczéma*, Darier) and general (*l'eczématose*, Darier). The latter group comprises eczema with a toxic anaphylactic or metabolic ætiology. In dealing with the metabolism of sugar in this condition, the author refers to true diabetes and to occult diabetes. He states that in the latter group dermatoses, and in particular eczema, are frequent complications. In this group, in which hyperglycæmia is present without glycosuria and acidosis, the skin lesions disappear after a strict alimentary *régime*, after reduction in weight and after insulin therapy. The author quotes two cases illustrating the use of blood sugar tests and the result of the adoption of a strict diet. The skin lesions disappeared with dieting and insulin treatment and no evidence of glycosuria was present.

Larva Migrans.

H. Montgomery (*Archives of Dermatology and Syphilology*, November, 1930) reports a case of *larva migrans* due to the larva of a botfly *gastrophilus*. The author demonstrated the larva in serial sections of the skin. The lesion appeared on a farmer on the internal surface of the right arm. The advance of the lesion amounted to 1·5 or 2·0 centimetres every day. When seen by the author, the area was eight centimetres in length and two millimetres in width. An elliptical area, two centimetres by one centimetre, was removed and the lesion disappeared. The author gives a detailed histo-pathological description of the condition and refers particularly to the difference between the effects of the presence of the organism itself and of toxins liberated by the larva and by scratching.

Centrifugal Erythema Annulare.

T. N. Graham and B. Throne (*Archives of Dermatology and Syphilology*, November, 1930) report in detail six cases of *erythema annulare* of the centrifugal type. This lesion is similar to that named by Calcott Fox *erythema figuratum perstans*. A typical lesion begins as a red papule which spreads peripherally to form a large circle of papules, leaving an area of clear or slightly discoloured skin in the centre. The pattern changes so rapidly that there may be a complete transformation in ten days. The disease may disappear after a few weeks and there may be rapid reappearances. There may or may not be desquamation or pruritus. Vesicles have been reported. The speed of evolution and the widespread distribution of the eruption distinguish it from *granuloma annulare*. In some instances the condition has been treated as syphilis. The annular variety of *erythema multiforme* is more acute and has a different type of evolution and involution.

UROLOGY.

Arteriography of the Kidneys.

R. dos Sartos (*Journal d'Urologie Médicale et Chirurgicale*, July, 1930) describes a method of diagnosis in renal and other upper abdominal lesions. Sodium iodide in very high concentration is injected into the abdominal aorta by a long needle introduced dorsally below the twelfth rib. The exposure is made at once. It is claimed that no cases of iodism have occurred among fifty patients, and that use of the arterial route causes less toxicity than the venous route. By this method it is possible to visualize radiographically the renal circulation in the living subject. The author and his collaborators consider themselves justified in pursuing their researches and they hope to reveal a new aspect of the pathological anatomy of the kidney by this means.

Ureteric Stricture.

S. Brügel and R. Paschkis (*Zeitschrift für Urologie*, February, 1930) have made a study of the ureter in human embryos and infantile cadavers. The whole of the retroperitoneal tissues were removed as quickly as possible after death and fixed in formalin. Later the whole ureter, together with the adherent connective tissue around it, was carefully prepared and sectioned. The authors sought to investigate the real incidence and the existence of pathological stricture of the ureter, as there seems to be serious doubt on this problem in the minds of many clinical workers. The researches showed that the muscular layer of the ureteric wall varies very greatly in different portions of the canal and that these variations are by no means constant in position or degree, even within the limits of what may be considered anatomical normality. Where the musculature is greatly developed, a definite narrowing of the ureteric lumen is visible to the naked eye, and often a thickening of the connective tissue outer sheath of the ureter is seen in conjunction with the muscular thickening. Most of these developments occur in the lower portion of the ureter and the thickenings did not correspond strictly in position to the areas commonly known as points of anatomical narrowing, but were met with at other points in the lower portion of the ureter. In none of these preparations were there any signs of inflammatory changes, so it must be acknowledged that many so-called "strictures," especially when diagnosed by the Hunner method, are

really physiological or anatomical variations in the canal. The authors quote other recent work which shows that the calibre of the normal ureter varies between that of an eight and of a twenty French catheter in different parts of the canal and that the commonest area of anatomical narrowing is in the lower four centimetres. It is to be noted that the great majority of "strictures," which are supposed to have been correctly diagnosed by modern methods, lie in this portion. The authors consider that the greatest care in diagnosis should be taken before a ureteric stricture, which should be a definite pathological process, is diagnosed.

Renal Functional Tests.

B. A. Thomas (*Journal of Urology*, August, 1930) reviews the chief kidney function tests in use at present and gives his opinion on their relative value. He thinks that undoubtedly the most reliable test is a quantitative metabolic study, such as the Mosenthal test day meal; this test is invaluable for the determination of incipient nephritis, but is a measure only of the lessening of versatility of kidney function in relative insufficiency and is of no value in absolute renal insufficiency. For the urologist, however, the tests of practical value fall into two groups, tests of retention and tests of excretion. The determination of the total non-protein nitrogen is one of the most dependable tests of nitrogen retention in the blood. The test for urea nitrogen is more simple and has replaced that of the non-protein nitrogen on that account, and, moreover, is equally dependable. The urea salivary index is a satisfactory guide and its findings run parallel to the blood urea results. The blood uric acid test is of doubtful value. In the group of tests of excretion, the estimation of the elimination of an endogenous product of metabolism, such as urea, is an ideal test and the urea concentration test of McLean is of great value. Various exogenous substances have been used in tests of excretion, but the most useful are test dyes, indigo-carmine and phenolsulphonephthalein. The latter is ideal when an actual quantitative estimation is required, but the former is a quick and practical test and is rendered of great value when the "index of elimination" is estimated; this index is defined as the ratio of the output of the first and third periods of the normal cycle of elimination. Normally the kidneys will eliminate about five times as much dye in the first as in the third period of twenty minutes following an intravenous injection of the dye.

Renal Sympathectomy.

E. Hess (*The Pennsylvania Medical Journal*, August, 1930) considers the clinical indications for performing denervation of the kidney pedicle, and describes the operation and its results. In the series of operations performed by the author the clinical indications were: (i) Nephralgia, (ii) nephralgia *plus* nephrosis, (iii) nephralgia *plus* nephrosis and ptosis, and (iv) as a preventive for the reformation of pelvic calculi. The operation is perfectly simple, provided the denervation is done delicately and slowly. The thin fascia overlying the arteries and veins is teased away with forceps and the nerve filaments are picked off the vessels by slipping a grooved director under them and then cutting on to the director with a fine knife. The proximal ends of the nerve filaments are then lightly brushed back medially with gauze towards the aorta and the *vena cava*. Associated with the nerve filaments in the fine fascia, which is then brushed back, are many lymphatic vessels. After the pedicle has been completely denuded, the renal pelvis is stripped with gauze and the ureter is separated from its bed and from its fascial attachments as far down as is possible; a light stripping of the ureter is all that is necessary. No attempt is made to suspend or fix the kidney in a higher position, even if ptosis is present and no decapsulation is done. A certain amount of fixation of the kidney results from post-operative adhesions. The results have been good and no apparent harm has resulted to the ureter, pelvis, calyces or kidney parenchyma. This has been proved by pyelographic and bilateral functional studies.

Cystitis Emphysematosa.

R. G. Mills (*The Journal of Urology*, September, 1930) reports a further case of *cystitis emphysematosa* in a male, which, he claims, is the sixth on record. Diagnosis was made *post mortem* and the pathology is dealt with in detail. The condition was associated with *cystitis cystica*, suggesting a preliminary inflammatory process. Perineal prostatectomy had been performed and the affected region was limited to the bladder base, the area liable to trauma during investigation and after treatment. It is doubtful whether the disease has ever been diagnosed clinically in the male and the author refers to the difficulty of cystoscopic differentiation from *cystitis granulosa* and *cystitis cystica*.

Spinal Anæsthesia for Genito-Urinary Operations.

M. F. Campbell (*The Journal of Urology*, September, 1930) advocates the use of spinal anæsthesia for genito-urinary surgical operation from the experience of 1,520 personal cases. He favours the Pitkin technique with preliminary morphine medication. Contraindications are: (i) Very young patients, (ii) extremely nervous patients, (iii) presence of spinal disease or brain tumours, (iv) cardiac or respiratory embarrassment. Moribund or extremely weak patients are better treated by other forms of regional anæsthesia. Hypotension, since the introduction of ephedrin, is a relative contraindication only. Failure to obtain satisfactory anæsthesia occurred in 2·9% of cases and was rectified by repetition of spinal anæsthetic, reinforcement by infiltration or general anæsthesia. Some failures are inexplicable; others are due to extradural injection or inert drugs. There were four deaths in the series, only one of which is attributed to the anæsthetic. This was in a case of *Bacillus coli communis* meningitis and was due to imperfect preparation rather than faulty methods. In regard to post-anæsthetic complications, headache occurred in 3%. The author finds it incurable, but self limited, the longest duration being ten days. It appears to be due to dural leakage. Respiratory embarrassment is avoided by maintenance of the blood pressure and by a modified Trendelenburg position. Nausea and vomiting are caused by traction on viscera. Intestinal distension is much more uncommon than after general anæsthesia; pulmonary complicatians are rare and renal complications absent. The ultimate post-operative mortality was 4·4% less than the mortality in a series of similar operations under general anæsthesia.

Secondary Ureterectomy for Pain.

Two cases are reported by L. Michelson (*California and Western Medicine*, October, 1930) in which pain recurred after nephrectomy. In spite of normal clinical, cystoscopic and radiological findings, secondary ureterectomy was performed with complete relief in both instances. The only pathological conditions noted in the excised ureters were round cell infiltration and œdema and polymorphonuclear infiltration. The author argues that pain referred from the ureter is not invariably due to tension, but may be of inflammatory origin, possibly referred to the adjacent peritoneum.

Gonococcus Bouillon Filtrate.

B. C. Corbus and V. J. O'Connor (*The Journal of Urology*, September, 1930) report on the clinical results of intradermic injection of "gonococcus bouillon filtrate," a substance allied to, but differing from Besredka's antivirus. Local treatment with the filtrate failed, but intradermic injection is giving favourable results and the investigation is to be continued.

Spartein Sulphate in Suppression of Urine.

E. H. Ochsner (*Urologic and Cutaneous Review*, October, 1930) advocates the use of spartein sulphate in post-operative anuria. The dose advised is 0·06 to 0·12 gramme (one to two grains) hypodermically every three to six hours. It appears to have no effect on suppression from other causes, but the author states that he has not lost a patient with post-operative suppression in twenty-five years.

Shield-Shaped Kidney.

A case of the rare condition known as "shield-shaped kidney" is reported by G. Pesroso (*The Journal of Urology*, October, 1930). This is the least common of the renal anomalies and it is most unusual for a pre-operative diagnosis to be made. The author considers the pyelograms and X ray renal outline in his case are quite conclusive.

Special Articles on Diagnosis.

(Contributed by Request.)

XXXIX.

PNEUMONIA.

A sudden onset with rigor and pain in the side of the chest, high fever from the beginning of the attack, painful, grunting, frequent respirations, with perhaps a short, unproductive cough: this combination of features makes the diagnosis of a typical attack of lobar pneumonia an easy one, even before the signs of consolidation of the lung or pleuritic friction can be discovered.

However, it is the purpose of this article to consider the diagnosis of the less characteristic forms of pneumonia, either occurring alone or as a complication of other diseases.

Apical Pneumonia.

When an upper lobe is affected, the initial features of the attack may be misleading. Pleuritic pain is often absent. I have noted several cases in which the respiration rate did not exceed 24 per minute, so that attention may not be specially drawn to the respiratory system. There is a tendency for the early onset of delirium, especially in children, and in them meningism is apt to occur.

The combination of delirium with retraction of the neck and absence (in the early stage) of abnormal physical signs in the chest indicate the necessity for diagnostic lumbar puncture. The withdrawal of a normal cerebrospinal fluid excludes the diagnosis of meningitis and a more careful physical examination may lead to the discovery of a high-pitched percussion note at one apex with distant tubular breathing and possibly fine, hair-like crepitations heard at the very end of inspiration over the affected area. In most cases these fine crackling crepitations are the earliest physical sign. One may hear them first high up in the axillary region and particular care should always be taken in the auscultation of this area. It is to be noted that in apical pneumonia the physical signs of consolidation are apt to appear later than in pneumonia of the lower lobe.

Meningism may also occur in basal pneumonia.

Pneumonia in Childhood.

In infants and young children lobular pneumonia may occur as a primary disease. It is, however, less common in its primary form than lobar pneumonia. Both forms are alike in their symptoms of onset. The attack is sudden and the child usually appears extremely ill from the first. A convulsion may be the initial symptom, representing the rigor of older patients. The child may lie on the affected side or be more comfortable propped up in bed. The *alæ nasi* expand with every inspiration. The respirations are very rapid in infants, sometimes reaching a rate of 60 or 70 or even more per minute. The respiratory rhythm is of the inverted type, that is, the pause follows inspiration instead of expiration. This is a valuable diagnostic sign. Physical examination is not likely to reveal much on the first day. After twenty-four hours, however, the characteristic fine crepitations may be heard over the affected area, which is usually at both bases in lobular pneumonia. In this form there are usually accompanying signs of bronchitis, namely, scattered râles, and sometimes, but not always, rhonchi.

Central Pneumonia.

Both in children and adults the pneumonic inflammation, beginning near the hilum of the lung, may spread to the pleural surface very slowly. This delays the appearance of the characteristic signs. Percussion may give a "boxy" or almost tympanitic note rather than flatness. Evidence of pleural friction is lacking and the respiratory murmur is apt at first to be diminished rather than tubular. As the inflammation reaches the surface, the more characteristic signs of consolidation appear.

Bronchopneumonia.

Bronchopneumonia occurs as a sequel to bronchitis from extension of the catarrhal process from the fine bronchioles into the alveoli. In childhood it is the commonest and most serious (in regard to total mortality) of the complications of measles and whooping cough. In adults it complicates many of the acute infections, especially influenza, though probably in regard to its pathology influenzal pneumonia should be placed in a group by itself. We find it also as an occasional complication of almost all general infections or asthenic types of illness.

The diagnosis of bronchopneumonia in adults seldom causes much difficulty. The patient's appearance suggests an acute respiratory illness. There are rapid respiration rate, cough and tenacious sputum which may be mucopurulent and perhaps blood-stained, but occasionally is absent. The lips, end of the nose, ears and finger tips may be cyanotic. The temperature is not often as high as in lobar pneumonia. The pulse varies with the height of the fever and the embarrassment of the circulation. The physical signs are those of basal consolidation, patchy or massive, on both sides, but often loud rhonchi render the auscultatory physical signs difficult to distinguish. If the affected lobules are patchy in distribution, percussion may not reveal very pronounced impairment of resonance, especially in fat people.

In small children the distinction between bronchopneumonia and bronchitis is notoriously difficult to determine by physical signs alone. One must consider the case as a whole. The respiratory distress is greater in bronchopneumonia, the respirations are more hurried, the *alæ nasi* work, the expiration is usually grunting and of the inverted type. The child's expression is usually either very anxious or unduly apathetic. The pulse rate is very rapid and signs of heart failure may appear early. This is shown by lividity, clammy, cold extremities and a tendency to *pulsus alternans*. (By this I mean a pulse in which, although the time rhythm is constant, some individual beats may be felt to be smaller than their fellows.) The temperature is higher in bronchopneumonia than in bronchitis and this has led to a generally accepted arbitrary rule, that a temperature of over 39·3° C. (102·8° F.) indicates that bronchopneumonia is present, as well as the more readily recognized bronchitis.

Catarrhal Pneumonia.

Catarrhal pneumonia is a synonym for bronchopneumonia, but I have used the term to denote a type of pneumonia of which I see some three or four cases every year. The onset as a rule is like that of influenza, that is, with malaise, pyrexia and general pains. Respiratory signs may not be prominent at first. There may be slight evidence of bronchial catarrh. The illness seems, however, more serious than ordinary bronchitis, and on the third or fourth day areas are discovered in one or both lungs, usually, but not always, towards the base, over which rather "sticky" medium-sized crepitations may be heard. There is not as a rule definite tubular breathing, nor is the percussion note much impaired. These areas may vary in position from day to day until defervescence. In some cases the physical signs become more definitely those of actual consolidation and the infection may run an ingravescent course with increasing alveolar involvement and eventually death. Most patients recover, however; in some of these during convalescence the crepitations persist. Not infrequently, if crepitations are heard at the apex of the lung and convalescence is slow, the physician may suspect the presence of apical tuberculosis. Several such cases of apical involvement persisting after influenzal pneumonia came under my notice after the great influenza epidemic of 1919. The patients all recovered in the course of a few weeks.

Tuberculosis and Pneumonia.

Sometimes pneumonia and pulmonary tuberculosis may be confused.

1. Pulmonary tuberculosis may be simulated by the catarrhal type of pneumonia described above.

2. Massive caseous pneumonia may be the first manifestation of pulmonary tuberculosis. This, however, is a

very rare condition. In the first few days of the attack the diagnosis of lobar pneumonia is certain to be made, but as the illness becomes protracted and there are high temperature, rapid wasting and no signs of resolution nor of a complicating empyema, the diagnosis of tuberculous pneumonia is suggested.

3. Bronchopneumonia in young children may run a very portracted, febrile course with much wasting, and this may cause suspicion in the mind of the physician that the condition is really one of tuberculous bronchopneumonia. But it should be remembered that tuberculosis of an acute pneumonic type is extremely rare in children. In these cases of bronchopneumonia the signs are basal and bilateral, vary from day to day and are usually accompanied by bronchitic rhonchi. On the other hand, when the lung is invaded by tuberculosis, the physical signs are more limited to a particular area and there is usually very little evidence of bronchitis. Unfortunately, in young children, sputum is not expectorated, but it may sometimes be recovered in the contents of the stomach after vomiting or aspiration, or the fæces may be examined for *Bacillus tuberculosis*. Bronchiectasis is a frequent sequel in these cases. Occasionally one sees protracted bronchopneumonia in adults, with pyrexia lasting for many weeks, in which tuberculosis may be suspected. In them, however, the signs are basal and usually bilateral and the sputum is persistently free from tubercle bacilli. Such conditions, as in children, may lead to bronchiectasis, with a persistence of signs at the base of one lung.

It should be remembered that subacute or chronic pulmonary infection of a purely basal type is almost certainly not tuberculosis.

Pneumonia and Bronchiectasis.

I remember being called in consultation to see a man very ill with acute lobar pneumonia at the base of the right lung. The point of interest in this case was that the illness had begun with a profuse hæmoptysis which was repeated on the third day. He undoubtedly had acute pneumonia, but the hæmoptysis was a puzzling feature. Inquiry elicited that since an attack of pneumonia complicating whooping cough in childhood he had a slight cough each morning and expectorated one or two fœtid sputa. In this instance the acute inflammation in the neighbourhood of a small bronchiectatic cavity must have caused hæmorrhage into the cavity, with the resultant hæmoptysis.

Bronchiectasis at the base of one or both lungs is not infrequently associated with recurring attacks of bronchopneumonia in the vicinity of the bronchial dilatations. The persistence of abnormal physical signs at the base of a lung in a child following an attack of bronchopneumonia should lead us to suspect the presence of such bronchiectatic changes. This brings us to the consideration of unresolved pneumonia.

Unresolved Pneumonia.

Unresolved pneumonia is rare after frank lobar pneumonia. In fact, one of the most striking pathological phenomena is the manner in which complete *restitutio ad integrum* occurs after even the most massive lobar consolidation. With bronchopneumonia, especially in children, it is otherwise. The products of inflammation may not be completely absorbed and the interstitial pulmonary and peribronchial tissue may become organized, leading to fibrosis. In such cases there may be a persistence of relative immobility, impaired resonance, râles and crepitations, indicating lessened aeration. Later, signs and symptoms of bronchiectasis may appear or be revealed by radiography following the instillation of "Lipiodol." It will be realized that the process is one of cicatrization rather than failure of resolution.

Occasionally so-called failure of resolution may be due to some more unusual and unsuspected cause. I recollect, many years ago, seeing a man who had been treated in a country hospital for pneumonia at the base of the left lung which had failed to resolve after defervescence. He had been discharged while he still had physical signs of impaired resonance, diminished breath sounds and distant crepitations at the lower posterior aspect of the left side of the chest. He obligingly made the diagnosis obvious by coughing up a large quantity of clear fluid and hydatid membrane a few minutes after I had finished examining him.

In all cases of unresolved pneumonia a very careful investigation should be carried out to ascertain the reason for the failure of resolution. The use of radiography is often essential.

Pneumonia Following Operation.

The symptoms and signs of pneumonia following operation do not differ from those of other forms of pneumonia, but the complication may be masked by the condition for which the operation was performed. A rising temperature and pulse rate, with undue hurrying of the respiration, will suggest the complication, which may be confirmed by physical examination. One must distinguish from pneumonia several other conditions: massive pulmonary collapse; pulmonary infarction; pulmonary abscess; pleural effusion, serous or purulent (empyema); hypostasis; subphrenic abscess.

Massive Collapse.

Although massive collapse more frequently follows abdominal operations near the diaphragm, it may occur after trauma, usually, but not invariably, of the chest wall. Many cases occurred during the war following gunshot wounds (not necessarily penetrating) of the chest. Reflex paralysis of some of the muscles of respiration has been regarded as its cause, but its pathology is obscure. The onset may be sudden, with symptoms and signs resembling pulmonary embolism. In other instances its onset is insidious and its presence revealed only by routine examination. Exertion, however, causes respiratory embarrassment and dyspnœa, rapid, shallow respirations, increased pulse rate, perhaps cyanosis and acute distress.

The physical signs are diminished respiratory movement on the affected side, with impaired percussion note; the heart is displaced to the affected side. The breath sounds may be absent, diminished or tubular. Adventitious sounds are absent at the onset. Later, the affected lung may expand to normal within two or three weeks and the signs disappear. Cough, with blood-stained sputum, may occur during this stage and adventitious sounds may be heard over the affected area. If the cause continues to act, pneumonia and pleural effusion may develop at the affected site and produce their own characteristic signs and febrile reaction.

Pulmonary Infarction.

The patient affected by pulmonary infarction may be in any stage of convalescence from an operation or at any stage of the puerperium. Sudden pain in one side of the chest, symptoms of shock and shallow, rapid respiration mark the onset. There may be a short cough with bloody sputum. After a day or so an area of consolidation may be discovered, perhaps associated with pleural friction over the affected area. The suddenness of onset, symptoms of collapse and slighter febrile reaction should make the diagnosis clear. In most cases, after two or three days the blood will give an indirect positive Van den Bergh reaction.

Pulmonary Abscess.

Insufflation during operation may cause pneumonia of the lobular type, usually basal and bilateral, or pulmonary abscess. Usually pulmonary abscess may be recognized by the more localized extent of the pulmonary signs and by the diminution of breath sounds. The expectoration of fœtid pus will confirm the diagnosis. Radiography, if available, may reveal a localized area of consolidation or possibly the abscess cavity. Operations on the mouth, that is, tooth extraction or tonsillectomy, are the most frequent causes of post-operative pulmonary abscess. In the former event it is not necessary for a portion of tooth or filling to be aspirated, for an abscess may follow aspiration of septic material which will not be revealed by X ray examination.

Pleural Effusion.

After operation pleural effusion (serous or purulent) may be mistaken for pneumonia.

A pleural effusion may be small in extent and the breath sounds may be tubular over the affected area and not notably diminished, but the symptoms are less acute. In many such cases, however, the use of an exploring needle may be required to settle the diagnosis. Of course, the two conditions (pneumonia and effusion) may coexist.

Subphrenic Abscess and Hepatic Abscess.

The temperature of a patient suffering from subphrenic or hepatic abscess is high usually, with morning remissions, and the patient is acutely ill. The upward bulge of the diaphragm may press upon and consolidate the overlying lung. The respiration rate is, however, seldom notably accelerated and the patient's appearance suggests the existence of sepsis rather than pneumonia. Not infrequently coarse friction sounds indicating an inflamed pleura are present; these may lead one to suppose that the lesion is primarily intrathoracic. Here again exploration is of great value, as the following history would indicate.

A man aged fifty years was seen nine days after operation at which a gangrenous appendix had been removed. A day or so after operation he complained of much pain in the lower part of the left side of the chest and the upper part of the abdomen; the pain was greatly increased by breathing. His temperature had remained between 37·8° and 39·5° C. (100° and 103° F.) after operation. Examination revealed diminished respiratory movement of the lower part of the left side of the thorax. Coarse friction sounds could be heard from the fourth rib downwards in the nipple line and in the left axillary region. There was a limited area of impaired resonance posteriorly, below the range of the left scapula, where the breath sounds were tubular, but diminished. Exploratory puncture in the dull area resulted in the withdrawal of a small quantity of offensive brownish fluid which contained broken-down red cells, pus cells and bacteria. At immediate operation an abscess was opened in the left hepatic lobe adjacent to its upper surface, below the left dome of the diaphragm. Brown offensive pus was evacuated.

Hypostatic Pneumonia.

Hypostatic pneumonia may occur as a complication in a variety of debilitating conditions, and is usually a terminal infection, especially in the aged. Its diagnosis is generally obvious. When an old or feeble subject in whom already signs of basal stasis and œdema have been discovered, becomes affected with increased rapidity of respiration, pyrexia and cough, with quickened, feeble pulse, together with increase of the consolidation and alveolar involvement, one may readily infer the existence of pneumonia and usually a fatal prognosis.

Abdominal Symptoms in Pneumonia.

In acute pneumonia of the lower lobe of the right lung the accompanying pleurisy may cause pain to be referred to the right lower quadrant of the abdomen, thus simulating acute appendicitis. However, the rapid respiration, the relative immobility of the lower part of the right side of the chest, and the absence of local tenderness should lead to a correct diagnosis if the possibility of referred pain is remembered. These cases occur most frequently in children.

In adults acute cholecystitis as well as appendicitis may be simulated, but in these cases also careful observation and evaluation of symptoms should enable their cause to be recognized.

C. T. CHAMPION DE CRESPIGNY, D.S.O., M.D. (Melbourne), F.R.C.P. (London),

Lecturer in Principles and Practice of Medicine, University of Adelaide; Honorary Physician, The Adelaide Hospital, Adelaide.

British Medical Association News.

ANNUAL MEETING.

THE ANNUAL MEETING OF THE NEW SOUTH WALES BRANCH OF THE BRITISH MEDICAL ASSOCIATION was held at the Robert H. Todd Assembly Hall, British Medical Association House, 135, Macquarie Street, Sydney, on March 19, 1931, DR. F. BROWN CRAIG, the Past President, in the chair.

ANNUAL REPORT OF THE COUNCIL.

The Honorary Secretary presented the annual report of the Council and moved that it be received. The motion was seconded by Dr. Kenneth Smith and carried. The report is as follows.

THE Council presents the following report on the work of the Branch for the year ended March 19, 1931.

Membership.

The membership of the Branch is now 1,684, as compared with 1,711 at the date of the last report, showing a net decrease of 27.

The additions have included elections and resumptions of membership, 70; removals into the area of the Branch, 36. The losses have included resignations, 19; removals out of the area of the Branch, 37; default in payment of subscription, 62; deaths, 15.

The losses by death have been: Dr. A. E. Brady, Dr. Cyril Shepherd, Dr. A. S. Vallack, Dr. R. L. Faithfull, Dr. R. W. Young, Dr. A. G. S. Gilchrist, Dr. J. K. Darton, Dr. C. S. Browne, Dr. A. Lewis Levy, Dr. C. V. Macinerney, Dr. G. P. Stanley, Dr. C. J. Peoples, Dr. K. M. Garrett, Dr. David Thomas, Dr. F. W. Raysmith.

Three extraordinary members were elected in accordance with By-Law 5.

Meetings.

Nine ordinary meetings of the Branch (including the Annual General Meeting), one extraordinary meeting and eight clinical meetings were held. The average attendance was 63. Six of the ordinary meetings, as follows, were held in conjunction with meetings of sections, namely: April 24, with the Section of Pædiatrics and the Section of Radiology; May 29, with the Section of Obstetrics and Gynæcology, the Section of Medicine, the Section of Neurology and Psychiatry and the Section of Hygiene and Preventive Medicine; June 26, with the Section of Pædiatrics, the Section of Orthopædics, the Section of Radiology and the Section of Pathology and Bacteriology; July 31, with the Section of Surgery, the Section of Medicine and the Section of Pathology and Bacteriology; August 28, with the Section of Medicine, the Section of Neurology and Psychiatry and the Section of Oto-Rhino-Laryngology; and November 27, with the Section of Pædiatrics, the Section of Pathology and Bacteriology and the Section of Medicine.

The clinical meetings were held at the Broughton Hall Psychiatric Clinic, the Royal Prince Alfred Hospital, the Royal North Shore Hospital, the Sydney Hospital, the Royal Alexandra Hospital for Children, Saint Vincent's Hospital, the Women's Hospital and Lewisham Hospital.

The business of the meetings during the year included twenty-one papers and addresses, numerous reports of cases, exhibits, and lantern slide and cinema film demonstrations.

Opportunity was taken at the ordinary meeting of October 20 to demonstrate fourteen lantern slides showing the British Medical Association House, 135-137, Macquarie Street, Sydney, and thirty-five slides, presented by the British Medical Association to the Branches in Australia, showing the British Medical Association House, Tavistock Square, London.

The Sydney University graduates (1930) in medicine were invited to attend the ordinary meeting of December 11, and the President welcomed them as members of the profession.

Representatives.

The Branch was represented as follows:

(*a*) *Council of the British Medical Association* (1930-1931): Professor R. J. A. Berry.

(*b*) *Representative Body* (1930-1931): Dr. R. J. Millard.

(*c*) *Federal Committee* (1930): Dr. J. A. Dick, C.M.G., Dr. R. H. Todd; (1931): Dr. J. A. Dick, C.M.G., Dr. R. H. Todd.

(*d*) *Australasian Medical Publishing Company, Limited*: Dr. George Armstrong, Dr. T. W. Lipscomb, Dr. F. P. Sandes.

(*e*) *Council of the Bush Nursing Association* (1930-1931): The President.

(*f*) *Council of the Royal Society for the Welfare of Mothers and Babies*: Dr. R. B. Wade and Dr. A. J. Gibson.

(*g*) *Board of Control of the Campaign against Tuberculosis*: Dr. S. A. Smith.

Council.

(*a*) The attendance of the members of the Council and of the standing committees was as set out in the table on the next page.

(*b*) Leave of absence was granted to Dr. R. J. Millard, Representative of the Branch at the Annual Representative Meeting (1930) in London, and to Dr. R. B. Wade, a Delegate from the Branch at the Annual Meeting (1930) in Winnipeg, Canada.

(*c*) The representatives of the Local Associations of Members, appointed on the invitation of the Council to attend the regular quarterly meetings of the Council, were as follows: Dr. J. M. Alcorn (Central Southern), Dr. K. S. Macarthur Brown (1930) and Dr. C. W. Whiting (1931) (Central Western), Dr. A. M. Gledden (City), Dr. Hugh Hunter (Eastern Suburbs), Dr. W. F. Simmons (Illawarra Suburbs), Dr. D. G. Hunter (Kuring-gai District), Dr. F. S. Stuckey (Northern District), Dr. N. J. Mackay (South Sydney), Dr. Brooke Moore (Western), Dr. A. M. McIntosh (Western Suburbs).

Library.

Dr. J. A. Dick was again appointed to the position of Honorary Librarian. Donations of books and periodicals were received from the Australasian Medical Publishing Company, Limited, the Public Library of New South Wales, the Medical School Library of the University of Otago, the American Association of Medical Milk Commissions, Dr. R. H. Todd, Dr. Garnet Halloran, Miss Faithfull (from the library of the late Dr. R. L. Faithfull), Sir Charles Clubbe, Dr. R. Cardamatis, Dr. W. H. Crago, Dr. J. Foreman, Dr. F. J. Williams, Dr. F. H. Vivian Voss, Dr. L. Cowlishaw, and Miss Margaret Windeyer. Dr. L. Cowlishaw has also given several interesting pictures.

Donations of journals were made to the library of the School of Public Health and Tropical Medicine, University of Sydney, and the Australasian Trained Nurses' Association.

Mrs. H. W. Armit presented a photograph of Lord Lister with signature.

The library has been much more used by Members since the Association moved into its new house. The *Complete Index to Surgery, Gynecology and Obstetrics*, Volumes I to IL, 1905 to 1925, has been purchased, and the *British Journal of Anæsthesia* has been added to the list of journals subscribed to. The Far Eastern Association of Tropical Medicine, Eighth Congress, Bangkok, December, 1930, was subscribed to for the purpose of obtaining the Proceedings of Congress.

Affiliated Local Associations of Members.

Balmain District (affiliated 1913): *Chairman*, Dr. H. K. Porter; *Honorary Secretary*, Dr. Roy Croft. Membership, 18. Three meetings were held.

Attendances at Council and at Standing Committee Meetings.

	Council.	Committees.					
		Executive and Finance.	Organization and Science.	Medical Politics.	Hospitals.	Ethics.	Post-Graduate Work.
Dr. J. E. V. Barling (Premises Attorney)	7	17	—	—	—	9	—
Dr. George Bell (President Elect)	6	19	7	12	6	7	7
Dr. C. B. Blackburn	5	—	—	—	—	10	—
Dr. A. J. Collins	7	—	10	—	5	—	—
Dr. W. H. Crago (Hon. Treasurer)	7	20	7	9	—	8	—
Dr. F. Brown Craig (Past President)	7	18	—	—	—	—	—
Dr. Andrew Davidson	6	—	—	11	—	10	—
Dr. A. M. Davidson	7	9	—	—	—	—	—
Dr. J. A. Dick (Hon. Librarian)	6	17	—	10	—	—	—
Dr. L. W. Dunlop	7	—	—	—	5	7	—
Dr. A. J. Gibson (Hon. Medical Secretary)	7	—	8	—	6	—	9
Dr. J. Hoets	6	—	8	11	—	—	—
Dr. E. M. Humphery (President [Resigned November, 1930])	4	13	3	6	1	5	4
Dr. C. H. E. Lawes	7	7	—	10	—	—	—
Dr. T. W. Lipscomb	6	14	—	12	—	—	—
Dr. R. J. Millard[1]	2	—	—	—	—	—	—
Dr. A. A. Palmer	7	—	—	—	—	10	—
Dr. Kenneth Smith	6	7	8	11	—	—	—
Dr. R. H. Todd (Hon. Secretary)	7	18	11	4	1	9	—
Dr. R. B. Wade[2]	2	7	—	—	3	—	—
Meetings held	7	20	11	12	6	11	

Dr. V. M. Coppleson[3]	3
Dr. W. Evans	7
Dr. W. W. Ingram[4]	2
Dr. E. H. M. Stephen	8
Dr. A. S. Walker	8
Meetings held	9

[1] Absent on leave March to December, 1930.
[2] Absent on leave July, 1930, to February, 1931.
[3] Resigned August, 1930.
[4] Absent on leave.

Border (affiliated 1908): *Honorary Secretary*, Dr. Affleck Robertson. Membership, 13.

Canterbury-Bankstown (formed 1930): *Chairman*, Dr. R. W. McCredie; *Honorary Secretary*, Dr. G. J. Cousins. Membership, 26. One meeting was held.

Central Northern (affiliated 1910): *Chairman*, Dr. J. B. F. McKenzie; *Honorary Secretary*, Dr. Alan T. Roberts. Membership, 74. Six meetings were held.

Central Southern (affiliated 1909): *Honorary Secretary*, Dr. S. P. Lyttle. Membership, 30.

Central Western (affiliated 1910): *Honorary Secretary*, Dr. K. S. Macarthur Brown. Membership, 59. Three meetings were held.

City (affiliated 1913): *Chairman*, Dr. Rees F. Llewellyn; *Honorary Secretary*, Dr. H. A. Ridler. Membership, 22. One meeting was held.

Eastern District (affiliated 1913): *Honorary Secretary*, Dr. R. A. Rankine. Membership, 11.

Eastern Suburbs (affiliated 1911): *Chairman*, Dr. Vivian Rich; *Honorary Secretary*, Dr. Hugh Hunter. Membership, 85. Six meetings were held.

Illawarra Suburbs (affiliated 1913): *Chairman*, Dr. K. G. Hearne; *Honorary Secretary*, Dr. G. F. L. Elliott. Membership, 37. Five meetings were held.

Kuring-gai District (formed 1929): *Chairman*, Dr. G. M. Hay; *Honorary Secretary*, Dr. F. A. E. Lawes. Membership, 43. Four meetings were held.

North-Eastern (affiliated 1913): *Chairman*, Dr. J. L. Roberts; *Honorary Secretary*, Dr. J. R. Ryan. Membership, 38. Three meetings were held.

Northern District (affiliated 1911): *Chairman*, Dr. R. B. Austin; *Honorary Secretary*, Dr. R. J. Jackson. Membership, 67. Three meetings were held.

South-Eastern (formed 1914): *Honorary Secretary*, Dr. C. P. Jackson. Membership, 10.

South Sydney (affiliated 1909): *Chairman*, Dr. T. K. Potts; *Honorary Secretary*, Dr. Justin Markell. Membership, 30. Four meetings were held.

Southern District (affiliated 1909): *Honorary Secretary*, Dr. C. R. Sim. Membership, 38.

Warringah District (formed 1929): *Honorary Secretary*, Dr. E. L. Newman. Membership, 54. Four meetings were held.

Western (affiliated 1908): *Chairman*, Dr. G. Fitz Hill; *Honorary Secretary*, Dr. S. R. Dawes. Membership, 80. Four meetings were held.

Western Suburbs (affiliated 1908): *Chairman*, Dr. W. M. Fletcher; *Honorary Secretary*, Dr. C. E. Vickery. Membership, 120. Three meetings were held.

The Canterbury-Bankstown Medical Association was formed, its area being the Municipalities of Canterbury and Bankstown, previously included in the area of the Western Suburbs Medical Association. The area of the Western Suburbs Medical Association has been amended accordingly.

The formation of the Kuring-gai District Medical Association and the Warringah District Medical Association, in 1929, to replace the Northern Suburbs Medical Association, has proved a convenience to the members in those localities.

Annual Meeting of Delegates.

The eighteenth annual meeting of delegates of the affiliated Local Associations of Members with the Council was held on October 3, 1930, in the Council Room, British Medical Association House, 135, Macquarie Street, Sydney. An account of the meeting appeared in The Medical Journal of Australia of November 29, 1930, at page 741, and a report of the proceedings was sent to the several local associations. The delegates present at the meeting were as follows: Dr. A. T. Roberts (Central Northern), Dr. G. A. Buchanan (Central Southern), Dr. C. W. Whiting (Central Western), Dr. A. M. Gledden (City), Dr. Hugh Hunter (Eastern Suburbs), Dr. W. F. Simmons (Illawarra Suburbs), Dr. L. Cowlishaw (Kuring-gai District), Dr. R. J. Jackson (Northern District), Dr. R. V. Graham (North-Eastern), Dr. H. H. Lee (South-Eastern), Dr. T. K. Potts (South Sydney), Dr. Murray Curtis (Warringah District), Dr. J. T. Paton (Western), Dr. W. M. A. Fletcher (Western Suburbs).

Sections for the Study of Special Branches of Medical Knowledge.

Genito-Urinary and Venereal Diseases (inaugurated 1928): *Honorary Secretary*, Dr. P. Fiaschi.

Hygiene and Preventive Medicine (inaugurated 1922): *Chairman*, Dr. Robert Dick; *Honorary Secretary*, Dr. A. H. Baldwin. Membership, 14. One meeting was held in conjunction with a meeting of the Branch.

Medical Literature and History (inaugurated 1925): *Chairman*, Professor Harvey Sutton; *Honorary Secretaries*, Dr. L. Cowlishaw and Dr. H. M. Moran. Membership, 30. One meeting was held.

Medicine (inaugurated 1924): *Chairman*, Professor C. G. Lambie; *Honorary Secretary*, Dr. E. H. Stokes. Membership, 35. Six meetings were held, including four in conjunction with meetings of the Branch.

Neurology and Psychiatry (inaugurated 1924): *Chairman*, Dr. N. D. Royle; *Vice-Chairman*, Dr. C. A. Hogg; *Honorary Secretary*, Dr. J. A. L. Wallace; *Honorary Medical Secretary*, Dr. R. A. Noble. Membership, 44. Six meetings were held, including three in conjunction with meetings of the Branch.

Obstetrics and Gynæcology (inaugurated 1925): *Chairman*, Dr. E. Ludowici; *Honorary Secretary*, Dr. H. A. Ridler. Membership, 33. Five meetings were held, including one in conjunction with a meeting of the Branch.

Orthopædics (inaugurated 1923): *Chairman*, Dr. H. R. G. Poate; *Honorary Secretary*, Dr. R. V. Graham. Membership, 21. Six meetings were held, including one in conjunction with a meeting of the Branch.

Oto-Rhino-Laryngology (inaugurated 1924): *Chairman*, Dr. Garnet Halloran; *Honorary Secretary*, Dr. Ashleigh Davy. Membership, 28. Four meetings were held, including one in conjunction with a meeting of the Branch.

Pædiatrics (inaugurated 1921): *Chairman*, Dr. Wilfred Vickers; *Honorary Secretaries*, Dr. F. C. Rogers and Dr. L. H. Hughes. Membership, 39. Seven meetings were held, including three in conjunction with meetings of the Branch.

Pathology and Bacteriology (inaugurated 1924): *Chairman*, Dr. A. J. Fitzgerald; *Honorary Secretary*, Dr. A. B. Lilley. Membership, 38. Six meetings were held, including three in conjunction with meetings of the Branch.

Radiology (inaugurated 1926): *Chairman*, Dr. W. B. Dight; *Honorary Secretary*, Dr. P. S. Parkinson. Membership, 22. Four meetings were held, including two in conjunction with meetings of the Branch.

Study of Cancer (inaugurated 1928).

Surgery (inaugurated 1925): *Chairman*, Dr. J. Colvin Storey; *Honorary Secretary*, Dr. C. E. Winston. Membership, 30. One meeting was held, in conjunction with a meeting of the Branch.

British Medical Association Lectures.

This system of lectures of a distinctive character on scientific and clinical subjects to be delivered at meetings of Local Associations of Members outside the metropolitan area was established in 1926.

Lectures were arranged as follows:

Central Southern Medical Association, Goulburn, March 14, 1930, Dr. Margaret Harper: "Infant Feeding."

North-Eastern Medical Association, Lismore, April 5, 1930, Dr. L. Gordon Tait: "Modern Views on Infant Feeding."

Northern District Medical Association, Narrabri, September 24, 1930, Dr. H. R. G. Poate: "The Diagnosis and Treatment of Pathological Conditions of Stomach, Duodenum and Gall Bladder."

Post-Graduate Work.

During the year several post-graduate courses were successfully organized and were well attended by Members.

From May 26 to June 6 a refresher course for general practitioners was held at the metropolitan hospitals, thirty graduates attending. Concurrently with this course a series of four lectures on "Structure and Function in Practical Medicine" was delivered by Professor F. Wood Jones, Professor of Anatomy, University of Melbourne. These lectures, at which ninety-five Members attended, were much appreciated by the audience.

Arrangements were made with the Melbourne Permanent Committee for Post-Graduate Work for Professor Evarts Graham, Professor of Surgery, Washington University, St. Louis, United States of America, to lecture in Sydney during his visit to Australia. Professor Graham delivered two lectures on the subject of "The Gall Bladder and its Disorders." One hundred Members attended these interesting and very instructive lectures.

In cooperation with the University Extension Board a course in radio-therapy was arranged. Sixteen Members attended the course, which commenced on June 17 and extended until the end of November.

Throughout the year regular weekly clinical demonstrations, open to all medical practitioners free of charge, were held at the metropolitan hospitals.

Federal Committee.

The Federal Committee of the British Medical Association in Australia met in Melbourne on March 28, 1930, and in Sydney on October 2, 1930. Reports of the proceedings of the Committee were published in THE MEDICAL JOURNAL OF AUSTRALIA of May 5, 1930, at page 630, and of November 22, 1930, at page 704, respectively.

Australasian Medical Congress (British Medical Association).

The fourth session of congress has been undertaken by the Western Australian Branch, to be held in Perth in September, 1932. Dr. D. D. Paton is the President and the Joint Honorary Secretaries are Dr. J. P. Ainslie and Dr. L. E. Le Souef, of Perth.

On the invitation of the Federal Committee and the General Committee of Congress, steps have been taken by the Council for the nomination of members in New South Wales as presidents and vice-presidents of sections of congress.

Dr. A. J. Gibson has accepted nomination as Local Secretary of Congress for New South Wales.

The Medical Journal of Australia.

In the last report the sad death after a short illness of the distinguished Editor, Dr. H. W. Armit, was referred to as having taken place on March 12, 1930. Dr. Mervyn Archdall, who had been Assistant Editor for some years, was at hand to carry on. He accepted the position of Editor and has filled the office with great ability. Dr. Archdall has for part of the year been ably assisted by Dr. W. L. Calov.

In addition to what it has done in recording the business meetings of the Branch and the annual and other meetings of the Local Associations of Members with the Council, the journal on its literary and scientific side has lived up to its high reputation. It has reflected the work done by the Branch during a year which has been a full one for papers and discussions and demonstrations at ordinary meetings, clinical meetings and meetings of the many sections for the study of special branches of medical knowledge.

The New House.

The Branch took possession of its beautiful new building, the British Medical Association House, 135-137, Macquarie Street, Sydney, at the end of June 1930, and at the beginning of July went into occupation of its offices, library, council chamber and committee room on the first floor. The assembly hall, which is on the ground floor, was used for the first time on May 28, when the first of a series of lectures by Professor F. Wood Jones, Professor of Anatomy, University of Melbourne, was given. The official opening of the new house took place on October 3, 1930.

It is to be deplored that the economic crisis which so suddenly overwhelmed the community after the middle of the year, should have made the immediate success of the house, as a financial undertaking, impossible. Despite the intention of many Members and others to make the house their place of practice, comparatively few only of the large number of suites of rooms have been taken, with the result that the incomings have been and are likely to continue to be insufficient to meet the necessary outgoings for some time to come. The Members have been appealed to to make it their business to endeavour to secure further tenants for the professional rooms; also to take up more debentures and each one to make a donation of £2 2s. to supplement the funds and so to tide over the period of shortage of revenue. Debentures (Series B) of £50, carrying interest at 6%, have been taken up to the amount of £23,130; and debentures (Series C) of £10, carrying the same rate of interest, to the amount of £2,340. Provision has been made, where so desired, for the debentures to be paid for by equal annual instalments over a period of five years. It is thought that this will make it easier for Members to take them up.

The building is all that it was hoped it would be, and, in addition to being beautiful within and without, it has afforded satisfaction to those who have made it their place of practice, and to the Branch as a whole, as a place of meeting.

Jubilee of the New South Wales Branch.

The Branch reached the fiftieth year of its establishment in 1930. The Jubilee celebrations were arranged to coincide with the official opening of its new home, the British Medical Association House, 135-137, Macquarie Street, Sydney. The opening ceremony took place on the afternoon of October 3, 1930, and was performed by His Excellency the Governor of New South Wales, Air Vice-Marshal Sir Philip Woolcott Game, G.B.E., K.C.B., D.S.O. An account of the proceedings was given in THE MEDICAL JOURNAL OF AUSTRALIA of October 25, 1930, at page 571.

The ceremony was followed in the evening by a dinner, at which His Excellency the Governor, Sir Philip Game, presided. The dinner was largely attended. The toast of the British Medical Association was proposed by Professor R. S. Wallace, Vice-Chancellor of the University of Sydney, in a brilliant speech, which was responded to in a characteristically charming speech by Dr. Robert Scot Skirving, to whom the profession in Australia owes so much as a leader of thought and as guide, philosopher, friend and champion.

Contract Practice.

The approved Common Form of Agreement for use between Medical Officer and Friendly Society Lodge has continued to work smoothly and with satisfaction. The number of Members undertaking friendly society lodge practice is 763. At the instance of the Friendly Societies' Association a conference with the Council was held on December 16, when a proposal of the Friendly Societies' Association in regard to medical benefits for lodge members who, owing to the financial depression, were out of employment and unable to pay their regular contributions, was discussed. The proposal has been submitted to the Local Associations of Members for consideration.

At the request of the Australian Natives' Association the question was reopened of extending recognition to it in connexion with contract medical benefit attendance on its members. It was decided that there should be no alteration in the existing relations with the Australian Natives' Association.

Workers' Compensation Act, 1926-29.

An increasing number of requests for advice in regard to attendance on injured workers has been received from medical attendants and insurers. This has added greatly to the work of the Medical Politics Committee and of the office generally.

At a meeting of the Local Associations of Members with the Council, held on April 24, 1930, it was resolved that a meeting should be arranged with insurers who are parties to the arrangement of October 4, 1927, in regard to amending Schedule "D." It was later decided, however, that the time for such a meeting was not opportune.

Certain insurers are contending that, as defined in the *Workers' Compensation Amendment Act* of 1929, hospital treatment includes medical treatment, and that, therefore, the employer is not liable for the cost of medical treatment, as distinct from hospital treatment, of an injured worker in hospital. A decision on this point by the Workers' Compensation Commission is necessary.

Public Hospitals Act, 1929.

A letter was received from the Hospitals Commission requesting that the Council would consider a proposal that the scale of fees chargeable by medical practitioners for attending intermediate patients in public hospitals should not exceed fifty *per centum* (50%) of the recognized scale of fees chargeable to full private patients. After communicating with the Local Associations of Members, it was decided to recommend to the Hospitals Commission that the fees chargeable by medical practitioners for attending intermediate patients in public hospitals should not exceed fifty *per centum* of the fees usually charged to private patients in the district in which the hospital is situated.

By-Laws—Advertisement.

Since the General Medical Council in June, 1923, for the purpose of enforcing, for the protection of the public, proper standards of conduct in medical practitioners in respect of advertising, decided to include in its "warning notice" a pronouncement prohibiting indirect advertising, the attention of the Council has been drawn from time to time to the necessity of amending the By-Laws of the Branch to bring them into accord with the rules of the profession as promulgated by the General Medical Council. The Council directed, therefore, that steps be taken for the adoption of the General Medical Council's rule governing direct and indirect advertisement as a By-Law of the Branch.

With the same object and in accordance also with a resolution passed at an extraordinary meeting of the Branch held on March 20, 1930, the Council has directed that steps be taken for deleting from the By-Laws the qualified sanction given in them for the advertisement in newspapers of commencement of practice and change of address.

Procedure in Ethical Matters.

The Central Ethical Committee of the British Medical Association has approved of the model rules governing procedure in ethical matters of a Branch in Australia, as adopted by the Federal Committee on April 10, 1929. The Council therefore has directed that these model rules be redrafted with a view to their being made By-Laws of the Branch.

Legislation.

The following Acts, passed during the year, are of special interest to medical practitioners:

(1) *Commonwealth Employees' Compensation Act.*

This Act provides, *inter alia*, that in addition to any compensation payable by the Commonwealth under this Act to or in respect of an employee, the Commonwealth Government shall pay the cost, not exceeding in any case the sum of one hundred pounds, of such medical, surgical and hospital treatment in relation to the injury as is, in the opinion of the Commissioner, reasonably necessary.

(2) *Opticians Act* (N.S.W.) 1930.

This Act provides for the registration and regulation of the practice of optometrists and for purposes connected therewith. It is noted that the provisions of the Act do not apply to any person registered as a legally qualified medical practitioner under the *Medical Practitioners Act* of 1912 or any Act amending or replacing the same.

F. Brown Craig,
Past President.

BRITISH MEDICAL ASSOCIATION—NEW SOUTH WALES BRANCH.

Receipts and Disbursements for the Year ended December 31, 1930.

Receipts.

	£	s.	d.
Balance at 1 January, 1930—			
Premises Account	4,500	0	0
Petty Cash	8	14	9
Subscriptions	8,320	15	6
Parent Association	4	17	6
Interest, Loan and Deposit	271	16	6
Sales, Lodge Agreement Forms	18	18	3
Rent, Sublettings	69	18	0
Balance, as per Cash Book	336	10	5
	£13,531	10	11

Disbursements.

	£	s.	d.
Debit Balance, as per Cash Book	23	19	11
British Medical Association	2,144	17	9
British Medical Association, Interest	10	14	6
The Medical Journal of Australia	2,094	8	9
Clerical Aid	2,789	5	0
Rent, Government Savings Bank	422	11	0
B.M.A. House	2,136	13	6
Printing and Stationery	240	14	2
Library	256	4	2
Sundries (including storage and removal)	181	16	0
New Chairs	49	0	0
Stamp Account	207	11	10
Federal Committee	171	10	0
Telephones	44	0	8
Jubilee Celebrations	39	19	9
Family Endowment Fund	20	5	9
Exchange	24	15	9
Expenses (B.M.A. Lectures)	26	11	0
Electricity and Gas	6	19	6
Balance at 31 December, 1930—			
Loan, Premises Account	2,635	3	0
Petty Cash	4	8	11
	£13,531	10	11

Examined and found correct.
A. Maitland Gledden
H. G. Humphries } *Auditors.*
March 5, 1931.

W. H. Crago,
Honorary Treasurer.

Premises Account.

Balance Sheet as at December 31, 1930.

Liabilities.	£ s. d.	£ s. d.
Debentures		27,790 0 0
Interest accrued on Debentures		1,587 11 8
Australian Mutual Provident Society	96,000 0 0	
Mortgage over Property, 135-137, Macquarie Street, Sydney—Interest accrued	526 13 4	
		96,526 13 4
British Medical Association, New South Wales Branch, Loan and Sundry Deposits		4,143 3 0
Sundry Creditors		56 3 8
Commercial Banking Company of Sydney, Limited		2,703 14 3
Accumulated Funds	55,995 10 10	
Less Deficiency in Revenue Account, 6½ months to 31 December, 1930	1,344 4 3	
		54,651 6 7
		£187,458 12 6

Assets.	£ s. d.
Land and Building, B.M.A. House, 135-137, Macquarie Street	185,120 13 0
Sundry Debtors	2,337 7 10
Cash on Hand	0 11 8
	£187,458 12 6

Revenue Account for Six and a Half Months ended December 31, 1930.

	£ s. d.	£ s. d.
To Sundry Expenses		2,419 18 6
„ Interest—		
Debentures	793 2 8	
Mortgage, etc.	3,733 10 7	
		4,526 13 3
		£6,946 11 9

	£ s. d.
By Rents earned	5,602 7 6
„ Balance—Deficiency for 6½ months to 31 December, 1930, transferred to Accumulated Funds Account	1,344 4 3
	£6,946 11 9

We have examined the books and vouchers of the Premises Account of the New South Wales Branch of the British Medical Association for the twelve months ended December 31, 1930, and we certify that in our opinion the attached Balance Sheet and accompanying schedules represent the true financial position of the Premises Account of the Association at December 31, 1930, and the transactions for the year ended that date respectively as shown by the books of the Association and information supplied us.

Coates, Cunningham & Co.,
Chartered Accountants (Aust.).

Financial Statements.

Dr. W. H. Crago, the Honorary Treasurer, moved that the statement of receipts and expenditure be received. The motion was seconded by Dr. J. E. V. Barling and carried. Dr. Crago also dealt with the balance sheet and financial statement of the premises account. In seconding Dr. Crago's motion that the statements be received, Dr. J. E. V. Barling explained the position. The motion was carried.

On the motion of Dr. Crago, seconded by Dr. T. W. Lipscomb, the thanks of the meeting were conveyed to Dr. A. M. Gledden and Dr. H. G. Humphries for their services as auditors.

Election of Office Bearers.

Dr. F. Brown Craig announced the result of the election of the President Elect and the members of the Council as follows:

President: Dr. George Bell.
President Elect: Dr. R. H. Todd.
Members of the Council: Dr. J. E. V. Barling, Dr. C. B. Blackburn, Dr. A. J. Collins, Dr. W. H. Crago, Dr. F. Brown Craig, Dr. A. M. Davidson, Dr. Lindsay Dey, Dr. J. Adam Dick, Dr. L. W. Dunlop, Dr. A. J. Gibson, Dr. A. W. Holmes à Court, Dr. C. H. E. Lawes, Dr. T. W. Lipscomb, Dr. R. J. Millard, Dr. A. A. Palmer, Dr. Kenneth Smith, Dr. R. B. Wade.

A vote of thanks was accorded to Dr. Garnet Halloran, Dr. H. S. Marsh, Dr. M. J. Plomley, Dr. H. A. Ridler, Dr. L. Gordon Tait, Dr. G. C. Willcocks, Dr. R. C. Winn and Dr. C. E. Winston for their services as scrutineers.

On the motion of Dr. W. H. Crago, seconded by Dr. A. A. Palmer, Dr. A. M. Gledden and Dr. H. G. Humphries were elected Honorary Auditors for the ensuing year.

On the motion of Dr. R. B. Wade, seconded by Dr. J. Adam Dick, Dr. R. H. Todd was appointed representative of the Branch in the Representative Body, 1931-1932.

On the motion of Dr. A. J. Gibson, seconded by Dr. J. Adam Dick, Dr. R. H. Todd was appointed delegate of the Branch to attend the annual meeting of the British Medical Association at Eastbourne. On the motion of Dr. Mervyn Fletcher, seconded by Dr. Kempson Maddox, Dr. Chester Dunstan was appointed second delegate to the annual meeting.

Incoming President's Address.

Dr. George Bell read his President's address (see page 395). A vote of thanks was passed to Dr. Bell on the motion of Dr. F. Brown Craig, seconded by Dr. R. B. Wade.

Induction of President.

In vacating the chair in favour of Dr. George Bell, Dr. F. Brown Craig said that Dr. Bell's year of office would not be an easy one. He knew that Dr. Bell would uphold the dignity of the office and hoped that he would have the support of members. The meeting closed with a vote of thanks to the Chairman, Dr. F. Brown Craig, moved by Dr. George Bell and seconded by Dr. R. J. Millard.

NOMINATIONS AND ELECTIONS.

THE undermentioned have been nominated for election as members of the New South Wales Branch of the British Medical Association:

Barry, Thomas, M.B., B.S., 1929 (Univ. Sydney), c.o. Dr. Fitzgerald, Newtown.
Donnellan, John Joseph, M.B., Ch.M., 1925 (Univ. Sydney), Dora Creek.
Jones, Karl Owen, M.B., B.S., 1928 (Univ. Sydney), "Mulwarree," Cordeaux Street, Campbelltown.

Correspondence.

WORKERS' COMPENSATION INSURANCE PRACTICE IN NEW SOUTH WALES.

SIR: The article on the above subject in THE MEDICAL JOURNAL OF AUSTRALIA of 14th instant, pages 211-214, written by Dr. J. G. Hunter, Medical Secretary, New South Wales Branch, British Medical Association, presents the subject very well from the point of view of the insurer. As in most instances, there is much to be said on both sides, and it is well that the view of the practitioner should be set out.

"Many of the disputes which have arisen between medical practitioners and insurers would have been avoided if the practitioners concerned had had a fuller understanding of certain sections of this act," says Dr. Hunter. Granted; but I believe that very many more disputes would have been avoided if the insurers concerned had had the honesty to stand up to the agreement entered into in Schedule "D." In this agreement the insurers undoubtedly gained an immense advantage. It is farcical to accept seven shillings and sixpence as the fee for a visit or consultation when the fee in private practice was ten shillings and sixpence and in some localities twelve shillings and sixpence. The mileage rate agreed on is even worse (better, of course, from the insurer's point of view). Between 8 a.m. and 8 p.m. we must accept five shillings per mile, when the rate in private practice is ten shillings and sixpence per mile, and between 8 p.m. and 8 a.m. seven shillings and sixpence per mile instead of twelve shillings and sixpence per mile. A footnote to this section of the schedule states that "four miles by rail is to be equivalent to one mile by road." Perhaps the insurer will pay only the rail rate when a train is available.

The sole compensating advantage we were to obtain through the adoption of Schedule "D" was to be the prompt payment of accounts. I think most practitioners can bear me out when I say that payment is not one whit prompter than before the agreement was made. As to the statement which is often made, that the practitioner is not obliged to work under Schedule "D," I should be very delighted and interested to hear of any payments being made under the terms of the pink schedule (Schedule "C" I suppose it was), when the insurers in many cases will not conform to the present one. These facts throw an interesting light on the words of Subsection 4 (*a*): "the customary charge made in the community for such treatment to persons other than workers."

On page 213 Dr. Hunter gives us some advice on the matter of cost of medical treatment. He there appears to set up two hypothetical cases of apparent overcharging and proceeds to demolish them. I do not wish to do him any injustice and would be glad to receive his statement that they were actual cases. If they were hypothetical cases he should realize that even the general practitioner would not accept the argument in these days. In place of his two cases I would place two cases which have come under my notice and are actual fact:

1. A practitioner attends daily from the eighth to the sixteenth inclusive an injured worker in hospital for a muscular and ligamentous sprain of the lumbar region of the spine, during which time he has occasion to needle the painful area twice. The worker is discharged from hospital and told to rest. He is seen on the third of the following month and discharged cured to his work. The account is sent in for £4 7s. The insurer writes: "In view of the nature of this man's injury the account appears to be a heavy one and the attendance frequent." The position is explained in writing. The insurer replies: "I am advised that a fee of £3 3s. would be commensurate with all the circumstances of this case and enclose herewith cheque for that amount."

2. A practitioner attends an injured worker on three successive days, once a day, for lacerated wounds of dirty fingers which it was thought inadvisable to suture. On the fifth day it was found that the wounds were very septic. The patient was not a fit case for hospital, but could not be trusted to dress himself properly. His hand was dressed on the fifth, sixth and seventh days. Not improving then, he was put in hospital and visited on the eighth, ninth, tenth, eleventh and thirteenth days. On the fourteenth day he was seen at the surgery and discharged to return to work in three days' time. Account for £4 12s. sent. The insurer replied: "Considering the nature of the injuries and the period that D. was incapacitated, we consider the number of attendances on D. far in excess of what they reasonably should have been, and as we are only liable for reasonable medical attention, we are reducing your account to £2 7s. and attach hereto our cheque for that amount, receipt of which kindly acknowledge." An explanation of the number of attendances was made, also a claim for the balance of the account and the wording of the letter strongly objected to. The insurer said: "We acknowledge receipt of your communication of the twenty-fifth instant in reply to ours of the twenty-first *idem* and fail to see that any exception can be taken to the tenor of the letter of that date." On being pressed to deal with the other points raised, the insurer did not reply.

Those two examples occurred in one man's practice in one month. There must be hundreds of them in this State during a year. The insurers not only have an immense concession in the rate of fees, but they are reducing the amounts still further in a totally unwarranted manner. The reference to Canadian and United States practice is wholly beside the point.

On page 214 reference is made to Subsection 6 (*b*). It is notorious that the insurer's medical officer under this section examines, treats and discharges worker patients without any reference whatever to the previous medical attendant.

A very curious sidelight on these points will be found in the Financial Supplement to *The Sydney Morning Herald* of thirteenth instant, page 1. Dealing with general insurance, it says: "The departmental returns following apply only to New South Wales, and are gleaned from figures supplied by the Government Statistician covering the twelve months ended June 30, 1930.

	Income.	Losses and Expenses.	Balance.
Fire	£2,377,300	£2,351,858	£25,442
Marine	427,263	332,233	95,030
Personal Accident	160,117	145,412	14,705
Workers' Compensation	1,532,671	1,318,426	214,245
Public Risk	49,101	31,908	17,193
Plate Glass	64,224	46,492	17,732
Motor Vehicle	1,090,971	1,000,220	90,751
Hailstone	54,576	40,736	13,840
Boiler Explosion	12,203	7,665	4,538
Live Stock	13,268	12,583	685
Burglary	59,212	47,563	11,649
Guarantee	33,216	36,193	– 2,977
Loss of Profits	64,624	41,509	23,115
Elevator	2,991	1,041	1,950
Sprinkler Leakage	2,832	1,903	929
Rainfall	6,085	4,758	1,327
Householder's Comprehensive	29,775	17,037	12,702
Other	3,578	4,019	– 441
	£5,984,007	£5,441,556	£542,451

From the above table it will be seen that the profits of workers' compensation insurance exceed those made under the next three most profitable heads combined, that they are about 40% of the total profits made by insurance

companies in New South Wales and that the rate of profit on workers' compensation insurance is about 14%. To anticipate critics I may state that I am aware that a "wise-crack" has it that statistics may be made to prove anything, even the truth. I do not want to suggest that these figures prove anything, not even that the insurers are making a lot of money out of the medical profession. I would, however, be extremely delighted to hear what Dr. Hunter thinks of them, if anything. They may even prove of interest to the general body of practitioners.

Yours, etc.,

J. R. RYAN.

Lismore,
February 15, 1931.

INFANT NUTRITION.

SIR: We read with interest the review appearing in your journal of 21st instant on Dr. McKim Marriott's work, "Infant Nutrition," and note the comparison in the calorific percentages of "Lactone" Syrup milk and Marriott's acid milk formula. Although this has little significance, we fear the comparison may bewilder the medical practitioner. Dr. Marriott recommends a reduction in the quantity of carbohydrate during the period of summer. The amount of sugar to be added to the lactic acid milk during this period is one ounce. The calorific percentages of this mixture and "Lactone" Syrup milk are as follows:

	Marriott's.	"Lactone" Syrup Milk.
Protein	16%	17%
Fat	36%	40%
Carbohydrate ..	48%	43%

We think this necessary adjustment escaped your notice. "Lactone" Syrup provides a convenient and safe method for the preparation of lactic acid milk; it can be made by any normal person, whereas for general use the lactic acid drop method is tedious by comparison. Moreover, the quantity measured in drops of lactic acid varies very greatly, as shown by the following figures:

10 drops lactic acid .. 3 minims (approximately)
20 drops lactic acid .. 6 minims
30 drops lactic acid .. 10 minims
40 drops lactic acid .. 13 minims
50 drops lactic acid .. 16 minims
60 drops lactic acid .. 20 minims

It is indeed difficult to conceive how an accurate quantity of lactic acid can be added to the milk, even by experienced mothers or nurses. Five years' practical experience of infant feeding with whole milk and "Lactone" Syrup has shown that it is a decided acquisition to the medical profession.

We have not advertised this product in the public press, and in Victoria we have reserved its sale to pharmacists. On all occasions we have willingly supplied full information regarding its composition and calóric value. These particulars are published in a "Guide to Infant Feeding," compiled by the Director of Infant Welfare in Victoria.

Since the introduction of "Lactone" Syrup we have received congratulations from numbers of medical men, many have expressed their appreciation of it, and now recognize that undiluted cow's milk to which it has been added, is of the utmost value as a well balanced nutritional diet for infants deprived of their natural food.

We agree with Marriott who says "that the final test of the adequacy of the diet is the response upon the part of the infant." Is there any other guide than this when an infant is breast fed? What further proof should be necessary when an infant is artificially fed? Since the essential requirement for successful artificial feeding is a well balanced diet, no artificial food is comparable with "Lactone" Syrup milk, which contains the requisite proportions of protein, carbohydrate, fats, mineral salts, vitamins and water. The child reared upon it obtains sufficient calories, and the food includes all the elements necessary for its welfare. Multitudes of milk mixtures when reduced to caloric percentages of their constituents may be almost identical except in so far as their concentration is concerned, that is, their water content. For example, it may be possible to have a mixture of very low calorific value containing small amounts of carbohydrates, fats and proteins, and yet the calorific percentages may be identical with a digestible mixture rich in proteins, fats and carbohydrates, and of a high caloric value.

The acidity of the gastric contents of an infant is the very foundation of Marriott's principle of artificial feeding with lactic acid milk, and its significance is well stressed in his original article published in the *American Journal of Diseases of Children*. The acidity of the gastric contents plays a very important part in the digestion of proteins as well as of fats. The value of lactic acid milk has been well established, particularly in the treatment of gastro-enteritis and summer diarrhœa. In no way does it usurp the natural function of the infant's digestive system, and consequently it can be fed to infants with great advantage up to twelve months of age, introducing the usual foodstuffs at the age of six months or thereabouts.

We have issued some literature in connexion with infant feeding, and we have pleasure in sending you two of the last pamphlets.

Yours, etc.,

L. R. SCAMMELL,
Managing Director,
F. H. Faulding & Company, Limited.

Adelaide,
February 27, 1931.

LOCAL ANÆSTHESIA.

SIR: One of the finest practical articles on the subject of local anæsthesia which it has been my fortune to read, is that which appeared in the journal of February 7 from the pen of Dr. Corlette. As one who has always been interested in this subject, I noted particularly the stress which the author laid on the five vital points: Premedication, gentle operating, suitable armamentarium, especially needles, safety of weak "Novocain" solutions, and minimum adrenalin content. Each of these points was dealt with adequately and from an intensely practical point of view. By his work in perfecting the morphine-hyoscine preparation of cases Dr. Corlette has carried out a piece of original research which ought find a place in future text books of practical pharmacology.

Dr. Corlette's advocacy of "Novocain" injections in the reduction of fractures coincides with my own much smaller experience in this work. Too often in the past fracture cases have had to wait until the anæsthetist has finished other work, but now it is practicable and safe to deal with such cases at once under local anæsthesia and, if skiagraphy shows poor position of the fracture, reduction can be repeated without further injection. I believe that in time this will become the routine procedure in all hospitals. As with all new methods, careful study is necessary in order to obtain the best results, and I would recommend those interested to read again Dr. Corlette's article, which presents the main points of Bohler's technique in brief and succinct fashion.

In conclusion, may I venture to hope that in the near future Dr. Corlette will give us a further article on this subject, which is of vital importance to all who have the best interests of surgery at heart.

Yours, etc.,

ROLAND A. H. FULTON.

2, Pitt Street,
Dunedin,
New Zealand,
March 11, 1931.

Obituary.

THOMAS O'LOGHLEN REYNOLDS.

DR. THOMAS O'LOGHLEN REYNOLDS died, as reported in a previous issue, on January 23, 1931, at Camberwell, Victoria. The son of Mr. Thomas O'Loghlen Reynolds,

Deputy Chairman of the Melbourne and Metropolitan Tramway Board, he was born at Coburg, Victoria, in 1899. He went to Saint Thomas's Grammar School and Scotch College, Melbourne. He became a medical student at the University of Melbourne in 1917. Ill health interfered with his studies and for this reason he did not graduate until March, 1930. He became Resident Medical Officer at the Toowoomba Hospital, Queensland, in May, 1930, but had to return to Melbourne owing to ill health in November, 1930.

Dr. S. V. Sewell writes:

Dr. Thomas O'Loghlen Reynolds was a delightful character, direct and full of humour and courage. In the second year of his medical course he developed pulmonary tuberculosis and from that time onwards his health was very indifferent and it was with difficulty that he completed his course owing to frequent relapses. It was my good fortune to look after him and his courage and splendid personality impressed themselves upon me.

The profession can ill afford to lose men of his type and character.

Books Received.

CANCER AND SCIENTIFIC RESEARCH, by B. Holmes, Ph.D., with a preface by F. G. Hopkins; 1931. London: The Sheldon Press. Crown 8vo., pp. 168. Price: 3s. 6d. net.

PRINCIPLES OF FUNCTIONAL ANATOMY OF THE RABBIT, by E. D. Crabb, Ph.D.; 1931. Philadelphia: P. Blakiston's Son and Company. Demy 8vo., pp. 144, with illustrations. Price: $1.50 net.

A HANDBOOK ON DISEASES OF CHILDREN, INCLUDING DIETETICS AND THE COMMON FEVERS, by B. Williamson, M.D., M.R.C.P.; 1931. Edinburgh: E. and S. Livingstone. Crown 8vo., pp. 302, with illustrations. Price: 10s. 6d. net.

DEAFNESS AND ITS ALLEVIATION BY OPERATION, by V. Nesfield, F.R.C.S.; Second Edition, 1931. London: H. K. Lewis and Company Limited. Demy 8vo., pp. 177, with illustrations. Price: 10s. 6d. net.

ANATOMY IN THE LIVING MODEL, A HANDBOOK FOR THE STUDY OF THE SURFACE, MOVEMENTS, AND MECHANICS OF THE HUMAN BODY AND FOR THE SURFACE PROJECTION OF THE VISCERA, ET CETERA, by D. Waterston; 1931. London: Hodder and Stoughton Limited. Royal 8vo., pp. 272, with illustrations. Price: 25s. net.

AN INTRODUCTION TO PRACTICAL BACTERIOLOGY: A GUIDE TO BACTERIOLOGICAL LABORATORY WORK, by T. J. Mackie, M.D., D.P.H., and J. E. McCartney, M.D., D.Sc.; Third Edition, 1931. Edinburgh: E. and S. Livingstone. Crown 8vo., pp. 435.

Diary for the Month.

Apr. 7.—New South Wales Branch, B.M.A.: Organization and Science Committee.
Apr. 9.—Victorian Branch, B.M.A.: Council.
Apr. 9.—New South Wales Branch, B.M.A.: Clinical Meeting.
Apr. 10.—Queensland Branch, B.M.A.: Branch.
Apr. 14.—New South Wales Branch, B.M.A.: Ethics Committee.
Apr. 21.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
Apr. 22.—Victorian Branch, B.M.A.: Council.
Apr. 24.—Queensland Branch, B.M.A.: Council.
Apr. 28.—New South Wales Branch, B.M.A.: Medical Politics Committee.
Apr. 30.—South Australian Branch, B.M.A.: Branch.
Apr. 30.—New South Wales Branch, B.M.A.: Branch.

Medical Appointments Vacant, etc.

For announcements of medical appointments vacant, assistants, *locum tenentes* sought, etc., see "Advertiser," page xviii.

Children's Hospital (Incorporated), Perth, Western Australia: Junior Resident Medical Officer.

Sydney Hospital, Sydney, New South Wales: Honorary Vacancies.

Medical Appointments: Important Notice.

Medical practitioners are requested not to apply for any appointment referred to in the following table, without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

BRANCH.	APPOINTMENTS.
New South Wales: Honorary Secretary, 135, Macquarie Street, Sydney.	Australian Natives' Association. Ashfield and District United Friendly Societies' Dispensary. Balmain United Friendly Societies' Dispensary. Friendly Society Lodges at Casino. Leichhardt and Petersham United Friendly Societies' Dispensary. Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney. North Sydney Friendly Societies' Dispensary Limited. People's Prudential Assurance Company, Limited. Phœnix Mutual Provident Society.
Victorian: Honorary Secretary, Medical Society Hall, East Melbourne.	All Institutes or Medical Dispensaries. Australian Prudential Association Proprietary, Limited. Mutual National Provident Club. National Provident Association. Hospital or other appointments outside Victoria.
Queensland: Honorary Secretary, B.M.A. Building, Adelaide Street, Brisbane.	Members desiring to accept appointment in ANY COUNTRY HOSPITAL, are advised to submit a copy of their agreement to the Council before signing, in their own interests. Brisbane Associated Friendly Societies' Medical Institute. Mount Isa Hospital. Mount Isa Mines.
South Australian: Secretary, 207, North Terrace, Adelaide.	All Lodge Appointments in South Australia. All Contract Practice Appointments in South Australia.
Western Australian: Honorary Secretary, 65, Saint George's Terrace, Perth.	All Contract Practice Appointments in Western Australia.
New Zealand (Wellington Division): Honorary Secretary, Wellington.	Friendly Society Lodges, Wellington, New Zealand.

Editorial Notices.

Manuscripts forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to The Medical Journal of Australia alone, unless the contrary be stated.

All communications should be addressed to "The Editor," The Medical Journal of Australia, The Printing House, Seamer Street, Glebe, New South Wales. (Telephones: MW 2651-2.)

Subscription Rates.—Medical students and others not receiving The Medical Journal of Australia in virtue of membership of the Branches of the British Medical Association in the Commonwealth can become subscribers to the journal by applying to the Manager or through the usual agents and booksellers. Subscriptions can commence at the beginning of any quarter and are renewable on December 31. The rates are £2 for Australia and £2 5s. abroad *per annum* payable in advance.